

# THE AMERICAN SURGEON

©Copyright 1960, The Williams & Wilkins Co

Vol. 26, No. 8

August, 1960

## AN ANALYSIS OF 1500 BREAST BIOPSIES

FRANCIS M. MASSIE, M.D.\* AND JAMES T. MCCLELLAN, M.D.†

*Lexington, Kentucky*

There have been many reports on the various phases of cancer of the breast. Previous studies have attempted to throw light on the relation between cancer and the so-called "precancerous" lesions: metaplasia, hyperplasia (both intra-ductal and alveolar), papilloma formation, and diffuse cystic disease ("chronic cystic mastitis"). The effects of different surgical attacks on cancer of the breast have been reported in great detail, most with "follow-up" studies covering 5 to 10 years or longer. These therapy reports have shown the results of radical surgery with and without x-ray therapy, simple mastectomy with x-ray therapy, extensive surgery of the supra-clavicular and mediastinal nodes, castration in premenopausal women, hormone therapy, and removal of adrenal and pituitary glands.

This report is based on a study of more than 1600 patients and 1589 biopsies during the years 1938 to 1959 inclusive (22 years) and will show: the incidence of cancer of the breast in a population of more than 130,000 seeking medical advice, the proportion of cancer in the "diagnostic" biopsy, the relation of routine physical examination to the finding of cancer unsuspected by the patient, the relation of pain to cancer, the incidence of other primary cancers in relation to the cancer rate in this age group, the incidence, not necessarily the relationship, of cancer to "aspiratable" cysts of the breast, and lastly on a study of sections made from breasts illustrating some of the points discussed.

During the period studied there were 135,997 admissions recorded including both sexes and

all ages. Some of these were admitted directly to hospitals, but most were first seen in the diagnostic offices of group practice. All of the surgery was done by 4 men and 50 per cent of it by one. In this series there were 619 cancers of the breast, or 4.6 per thousand, compared to the figures 1.62 per thousand national average. (table 1).

This is even more significant when we see that 342 of our total of 1630 were diagnosed before biopsy as cancer and 54 or 16 per cent of these were wrong. How serious it would have been to do radical surgery on these!

On the other hand, 501 patients had lesions we thought quite surely were benign and 18 of these were cancer or 3.75 per cent. The margin of possible error is even greater in those patients about whom we were doubtful. There were 746 of these, of whom 117 had cancer. Of these, cancer was thought likely in 63 and unlikely in 54.

If these figures mean anything, and we are sure they do, they mean the only way to be certain is to biopsy *all* breast tumors. The policy of "wait and see" is never justifiable (except in patients under 20 years of age). Wait and see what? Until the diagnosis can be made on physical examination? Our experience (as above) shows that this is not reliable without biopsy.

We think it is important to note the lowering percentage of cancer in our "biopsy" patients over the years (table 2). In our first 8 years of 350 biopsies only 150 were benign or 57 per cent cancer. The next 7 years brought 505 biopsies with 305 of them benign or 40 per cent cancer; the final 7 years of this study showed 775 biopsies and 556 benign or 28 per cent cancer. We believe whenever there is an incidence of more than 40

\* From the Department of Surgery, Lexington Clinic, Lexington, Kentucky.

† From the Department of Pathology, Lexington Clinic, Lexington, Kentucky.

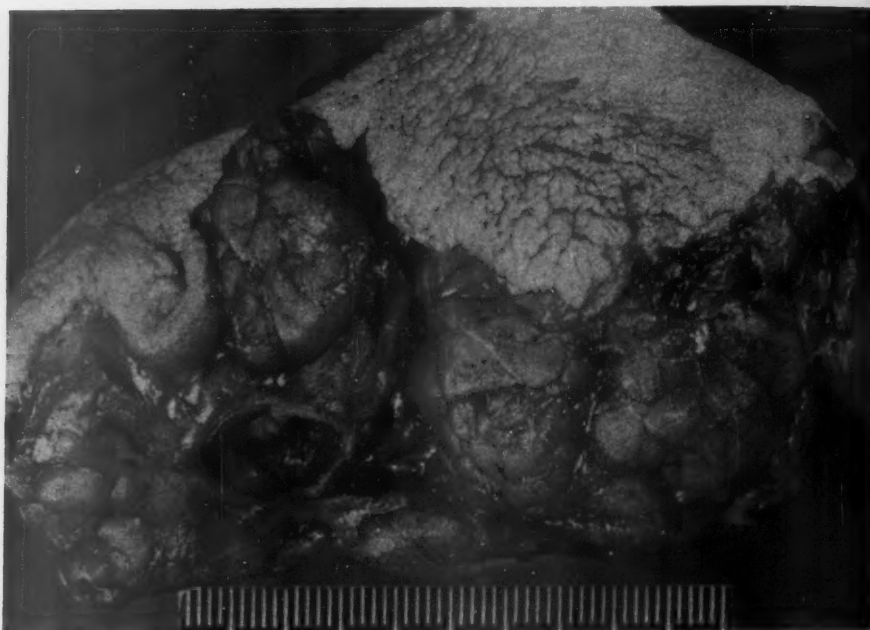


FIG. 1. Section of breast showing dilated duct with intraductal papilloma

per cent cancer in breast biopsies that not enough biopsies are being done.

The value of routine complete physical examination is well illustrated by our figures of 15 breast cancers (in 619 patients) where breast lesions were not suspected by the patient or 2.4 per cent of our cancers. We found no relation between the extent of the disease and the history of duration before seeking medical advice. This can be explained in two ways and probably both are partly right: (1) the patient is ashamed to say how long she has had a "lump" in the breast, has concealed it and does not tell the truth when she finally consults a doctor; and (2) far more important, she has not discovered the "lump" until it is advanced. The only answer to late discovery is to teach women to examine their breasts with intelligence and regularity.

We have often heard that pain in the breast means "no cancer." With this comforting and fallacious statement we cannot agree. We have observed 476 patients over 40 years of age who have given a history of localized pain in a breast. We have considered only those over 40. The complaint is common and, in the absence of a lump, may be safely watched below that age.

TABLE 1

*Carcinoma of the breast 1938 to 1959*

Total patients seen.....	135,997
Cancer of breast.....	619
No operation.....	6
No biopsy.....	35
Biopsy.....	578

TABLE 2

*Cancer rate expressed as the percentage of biopsies*

Years	No. of Biopsies	Benign	Cancer	
			no.	%
1938 to 1945	350	150	200	57
1946 to 1952	505	305	200	40
1953 to 1959	775	556	219	28

We have excluded all those with diffuse pain. Of these 476 who had local pain and tenderness in one breast, 119 had cancer. Of every 4 women over 40 years old who come to a doctor complaining of local breast pain, one will have cancer.

In our 619 patients with cancer of the breast 17 or 2.7 per cent, which is 27 per thousand, had

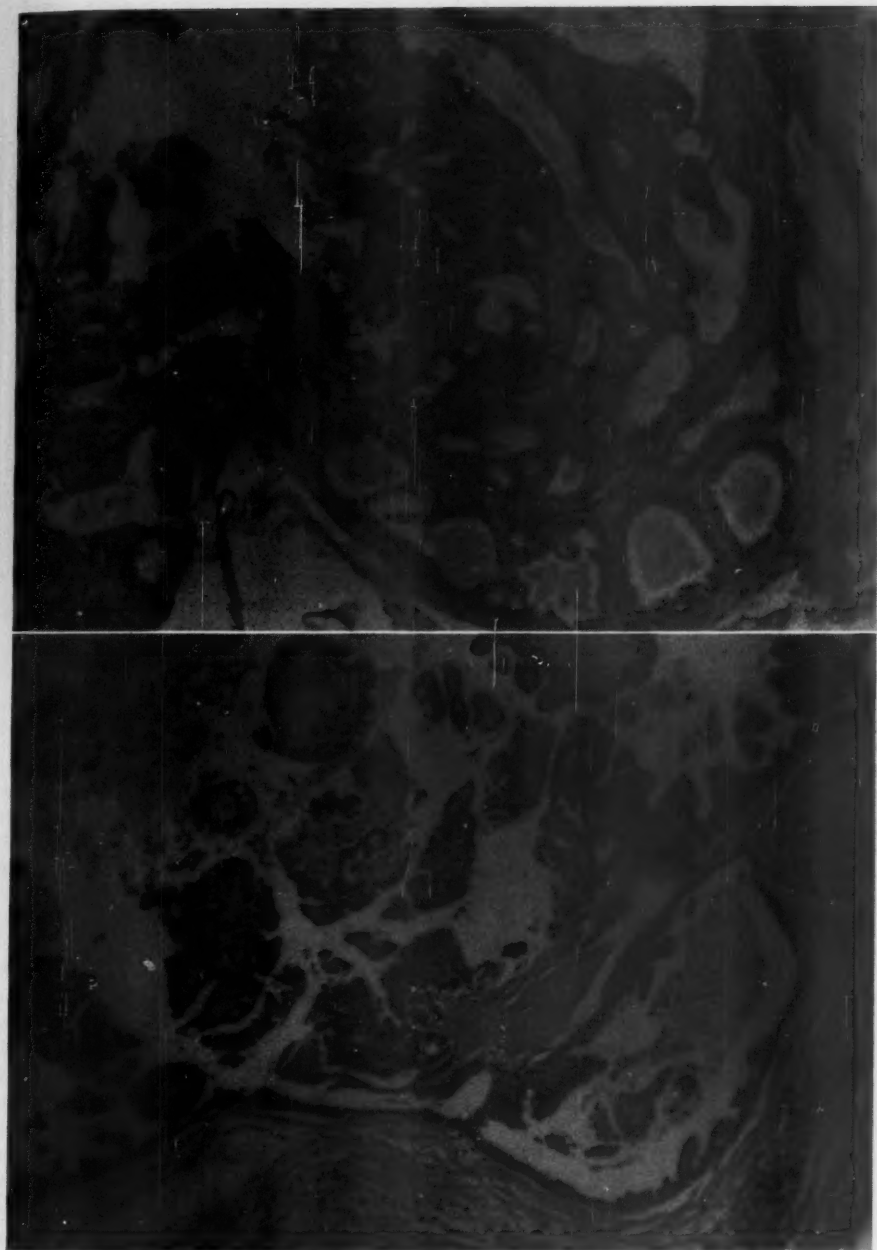


FIG. 2. *Upper.* Mammary duct in breast tissue shown in figure 1 showing benign intraductal papilloma ( $\times 64$ ). *Lower.* Early papillary adenocarcinoma in duct with areas of possible invasion ( $\times 64$ ).

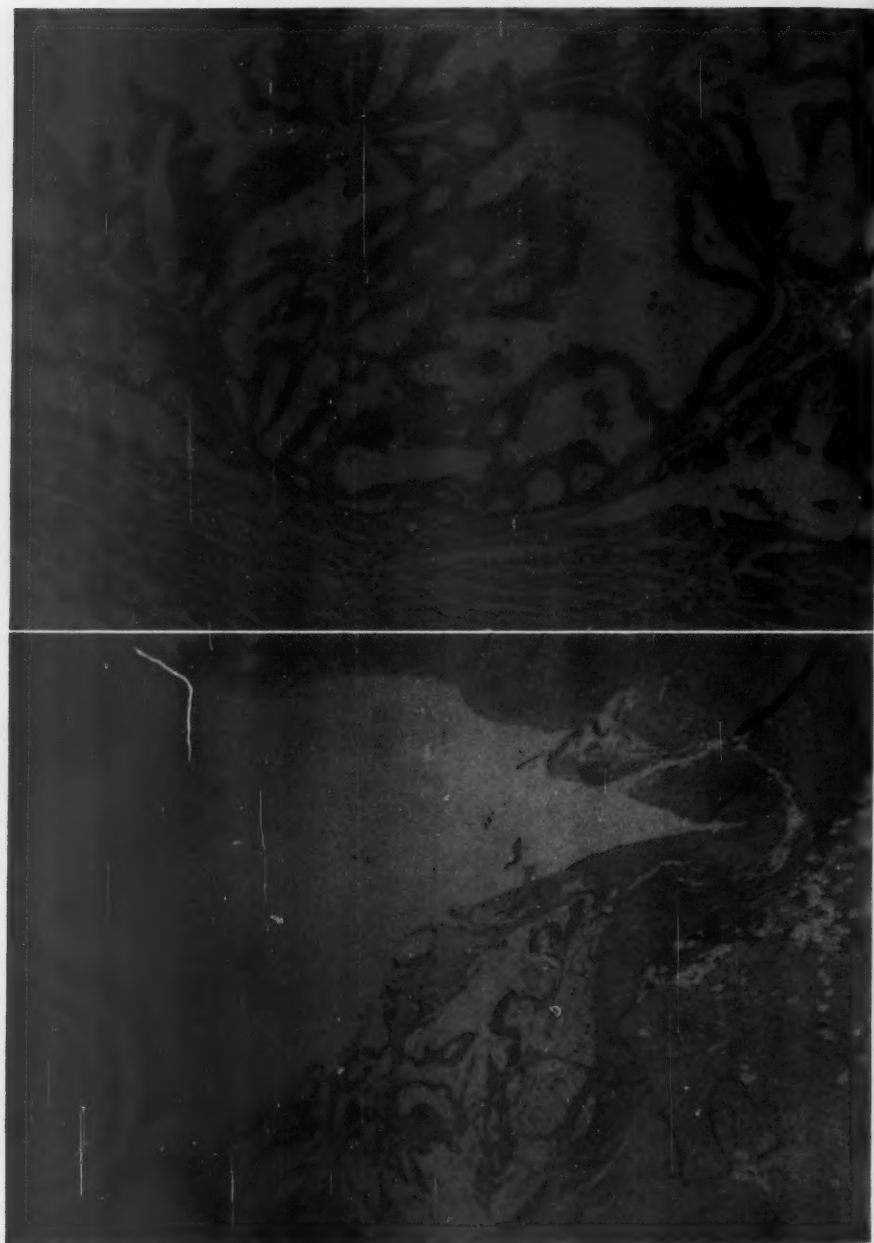


FIG. 3. *Upper.* Papillary adenocarcinoma in cyst ( $\times 64$ ). *Lower.* Infiltrating mucus-producing adenocarcinoma in wall of cyst ( $\times 16$ ).

other p  
immedi  
more th  
when su  
meaning  
in these  
pointing  
Some  
of the  
frequen  
show th  
the mos  
the fluid  
often in  
the cyto  
repeated  
dominant  
peritone  
cancer  
normal  
likely t  
steady.

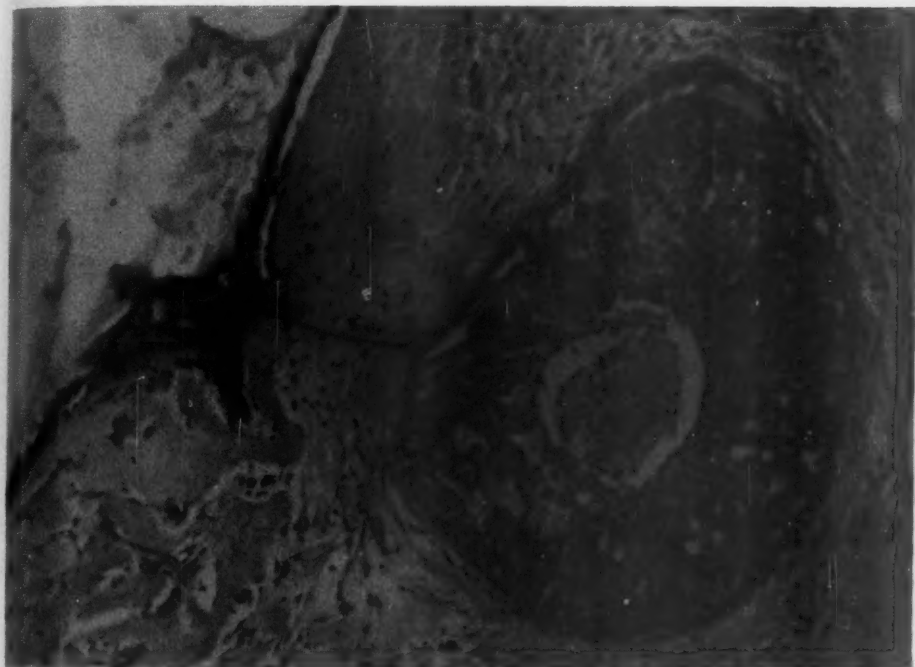


FIG. 4. Cystic disease in breast with intraductal carcinoma adjacent to cyst (X 68)

other primary cancers, usually in the 10 years immediately following. Probably there were more than this as some must have gone elsewhere when subsequent cancers developed. For us the meaning of this is: look for other primary cancers in these patients regardless of the lack of sign-pointing symptoms.

Some excellent surgeons still aspirate cysts of the breast either in the hospital or (more frequently) in the office. We think our figures show that this is a dangerous procedure even in the most experienced hands. The examination of the fluid so obtained for cancer cells results too often in false negatives. We have no figures on the cytology of aspirations but we remember the repeated negative reports on chest and abdominal fluid in known cases of pleural and peritoneal carcinomatosis. We suspect that any cancer which does not originate in tissue which normally exfoliates (squamous epithelium) is likely to produce a negative report on cytology study. The percentage of false negative cytologic

TABLE 3  
Cysts\* and cancer

	Cysts	
	no.	%
Benign.....	201	94.6
Cancer.....		5.5
Cyst wall.....	9	
Adjacent.....	2	

\* Cysts 1 cm. or more in diameter.

studies in cancer of the fundus, for example, is higher than in cancer of the cervix.

Our studies show (table 3) that we have had 212 patients with cysts of the breast 1 cm. or more in diameter. These we have classed as "aspiratable" cysts. We have purposely excluded reference to diffuse cystic disease. The relation of "chronic cystic mastitis" to cancer is still controversial. However, we regard this as a precancerous lesion. Of our 212, 201 were benign



FIG. 5. *Upper.* Infiltrating adenocarcinoma in breast tissue adjacent to cyst ( $\times 64$ ). *Lower.* Abscess in breast with infiltrating carcinoma ( $\times 64$ ).

(94.6 p  
arising  
adjacent  
cent of  
to igno

We  
other 8  
an absce  
of the  
found i  
suspect

1. Si  
cancer  
analyze
2. O  
benign,  
sies hav
3. In

(94.6 per cent) but 11 (5.4 per cent) had cancer arising from the cyst wall (9) or immediately adjacent to the cyst (2). We think the 5.4 per cent of breast cysts which are cancer is too great to ignore by aspiration.

We have found two patients, one 63 and the other 84 years old, who had cancer in the wall of an abscess. This serves to remind us that abscess of the breast is a disease of young women. When found in the elderly or aged it should make us suspect a causative or co-existing cancer.

#### SUMMARY

1. Six hundred and nineteen cases of breast cancer in a 22-year period have been partly analyzed.

2. Of our 1589 biopsies 67 per cent have been benign, but 84 per cent of 746 "diagnostic" biopsies have been benign.

3. In 117 cancers diagnosed by biopsy, 54

were thought probably to be benign and 63 probably malignant.

4. Routine physical examination will sometimes reveal cancer where the patient suspects no breast lesion. The extent (stage) of the cancer bears no relation to the history. Our greatest hope for early diagnosis is to train women to examine themselves with regularity, care and intelligence.

5. Cancer occurred in 25 per cent of localized pain in the breasts in the women over 40 in our series. This contradicts the comfortable but fallacious maxim: "Pain means no cancer."

6. The incidence of other primary cancer is three times the rate for cancer in this age group.

7. Aspiration without biopsy of breast cysts is dangerous because 5.4 per cent will show cancer in the wall of the cyst.

*Dr. Francis M. Massie*  
1221 S. Broadway  
Lexington, Kentucky

## A STATISTICAL ANALYSIS OF CHOLECYSTECTOMIES IN A CHARITY HOSPITAL

ROBERT O'NEAL CHADWICK, M.D.\*

Shreveport, Louisiana

The source of this information is a 1000-bed state owned completely charitable institution in Shreveport, Louisiana, dedicated to serve the indigent citizens of the state. The study includes all cholecystectomies done during the 5-year period from 1953 through 1957. Cholecystostomies were excluded as were cholecystectomies that were done in conjunction with radical surgical procedures (*e.g.*, hepatectomy).

From 1953 through 1957 there were 345 cholecystectomies performed; 312 (90 per cent) were elective and 33 (10 per cent) were emergencies; there were 286 women and 59 men operated upon for a ratio of 5 to 1. These figures are in accord with those published by Orr<sup>3</sup> and Evans.<sup>2</sup> Of the 345 patients 224 were Caucasian and 121 were Negro which is an absolute ratio of almost 2 to 1. When one considers the high percentage of Negro patients in our institution (ratio 2 to 1), the frequency of this disease in the Caucasian race compared to the Negro race becomes apparent. This is substantiated by the literature, but I can find no explanation for it.

The average age at the time of surgery was 55 years. For female patients alone the average age was 53.2 and for male patients, 65.2. This is one reason why the mortality rate in men is higher than in females. These average ages are higher than those published by Orr in his survey of 558 cases where he found the average age for women was 48 and for men 52; however, the consistency in older aged men is noted.

A total of 327 patients (95 per cent) had cholelithiasis and 51 (14.7 per cent) had choledocholithiasis. The calculi were radiolucent in 70 per cent and radio-opaque in 30 per cent. At some time 70 patients had been jaundiced, although only 57 of these had common duct explorations. Only 2 patients denied any abdominal or gastrointestinal symptoms and, in these, the stones were discovered at laparotomy for some other reason (table 1).

\* Resident, Department of Surgery, Confederate Memorial Medical Center, Shreveport, Louisiana.

Of the abnormal cholecystograms reported 48 per cent were nonfunctioning and 52 per cent were functioning and demonstrated stones.

There were 128 common duct explorations; this represents 37 per cent of the total. Of these, 51 patients or 40 per cent of those explored, had common duct stones. Approximately 14.7 per cent of the 345 patients had common duct stones.

At the time of admission 70 patients (20.3 per cent) were jaundiced or gave a history of jaundice; 57 of these patients had choledochotomies done and 29 (50.9 per cent) had common duct calculi. Of the 51 patients who had common duct stones 29 (57 per cent) had at some time been jaundiced.

Our common duct exploration rate of 37 per cent is higher than Thompson's<sup>4</sup> reported rate of 25 per cent on 500 cases in a community hospital, and considerably higher than Evans' 14 per cent in his study on private cases. Orr reports 38 per cent in 558 cases at the University of Kansas Hospital (table 2).

The average hospitalization time postoperatively when cholecystectomy and choledochotomy were done was 16.5 days, and when cholecystectomy alone was done, the average postoperative hospitalization period was 10.5 days.

There were 11 deaths for an over-all mortality rate of 3.2 per cent. In the elective cases alone there were 7 deaths for a mortality of 2.2 per cent, and when the elective procedures are considered exclusive of those where common duct exploration was done the mortality rate falls to 1.6 per cent (table 3).

As would be expected, the mortality rises sharply when the emergency procedures are investigated. In the 33 emergencies, there were 4 deaths for a mortality of 12.2 per cent, and when common duct exploration was done with emergency cholecystectomy, the mortality rate rose to an alarming 28.6 per cent (table 4).

These figures compare satisfactorily with those published in the literature. Thompson in 1951 reported a 3.8 per cent mortality in 500 cases

**TABLE 1**  
*Frequency of calculi, jaundice, and gastrointestinal symptoms*

	No. of Cases	Percentage
Cholecystectomies.....	345	100
Patients with cholelithiasis.....	327	95
Patients with choledocholithiasis.....	51	14.7
Patients with jaundice.....	70	20.3
Patients with gastrointestinal complaints.....	343	99

**TABLE 2**  
*Comparison of choledochotomies*

Studies	No. of Cases	Rate of CDE†	Positive CDE	CD‡ Stones in All Cases
		%	%	%
C.M.M.C.*.....	345	37	40	15
Thompson.....	500	25	47	12
Evans.....	275	14	25	4
Orr.....	558	38	34	13

\* Confederate Memorial, Medical Center.

† Common duct exploration.

‡ Common duct.

**TABLE 3**  
*Mortality in elective cases*

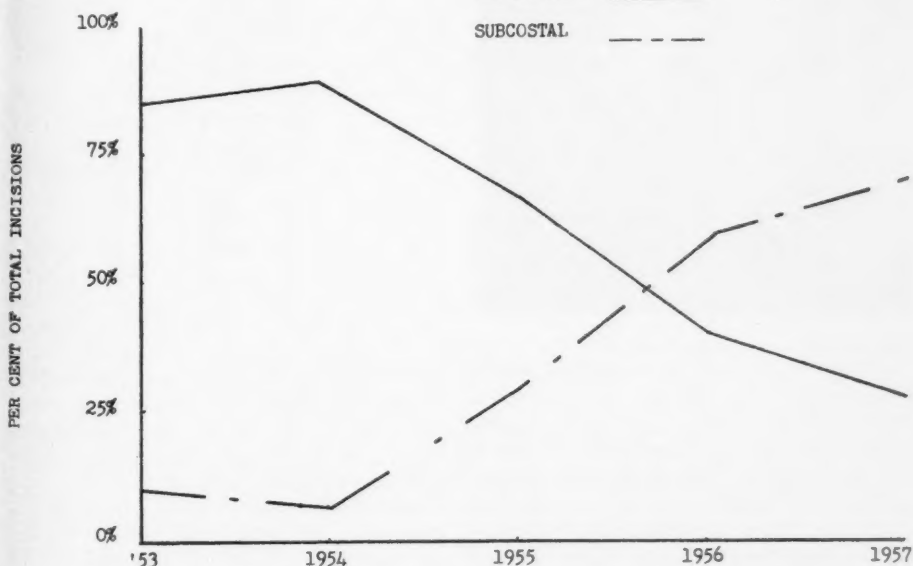
Operative Procedure	No. of Procedures	Deaths	
		no.	%
All cholecystectomies.....	345	11	3.2
Elective cholecystectomy without CDE.....	191	3	1.6
Elective cholecystectomy with CDE.....	121	4	3.3
All elective operations.....	312	7	2.2

**TABLE 4**  
*Mortality in emergency cases*

Operative Procedure	No. of Procedures	Deaths	
		no.	%
All cholecystectomies.....	345	11	3.2
Emergency cholecystectomy without CDE.....	26	2	7.7
Emergency cholecystectomy with CDE.....	7	2	28.6
All emergency operations.....	33	4	12.1

PARAMEDIAN

SUBCOSTAL



GRAPH COMPARISON OF THE FREQUENCY OF OPERATIVE APPROACHES

Fig. 1

done at a community hospital, and Evans in 1956 gave a 2.7 per cent mortality rate in 276 private cases. This lower rate is to be expected and is not out of proportion since it deals with private care in private hospitals. The overall mortality rate in men was 8.5 per cent and in women 2 per cent. Orr, in his study of 558 cases reported a mortality rate in men of 7.5 per cent and in women 1.8 per cent. The average age at death was 63.6 years. An interesting figure, and one which seems high today, is that published by Cole<sup>1</sup> in 1939, when he reported the mortality of operation upon the gallbladder as computed from numerous reports was about 6.5 per cent; he further stated that choledochostomy throughout the country probably carried not less than a 10 per cent mortality.

There were 133 subcostal or transverse incisions, 202 paramedian incisions, and 3 hockey

stick (Kehr) incisions. The remaining 7 surgical approaches were not specified in the charts. It is interesting to note graphically the increase in the popularity of the subcostal or transverse incision. When the percentage per year of each incision is thus plotted, this becomes obvious.

*Confederate Memorial Medical Center  
Shreveport, Louisiana*

#### REFERENCES

1. COLE, W. H.: Factors in the prognosis and mortality of gallbladder disease. *Surg. Gynec. & Obst. (Internat. Abstr. Surg.)*, 69: 40, 1939.
2. EVANS, A. L., COFER, O. S., GREGORY, H. H., AND CALK, G. L.: Biliary surgery. *J. M. A. Georgia*, 45: 519, 1956.
3. ORR, T. G.: A study of the mortality rate in a series of cholecystectomies. *Am. J. Digest. Dis.*, 14: 89, 1947.
4. THOMPSON, R. H.: Gallbladder surgery in a community hospital. *New England J. Med.*, 244: 347, 1951.

The c  
acrylic p  
since 19  
present  
cases. V  
regardin  
plasty, b  
Plasti  
for a nu  
have ut  
arthritic  
satisfact  
Reitz  
first to  
plastic.  
pioneers  
cranial  
Blaine  
which h  
in situ?  
been fol  
method  
and pre  
lumbia  
has bee  
of crani  
describi  
the Ma  
work u  
adopted  
experien  
Their p  
repair o  
present  
of repai  
methac  
been en  
Spence  
plasty p  
over a  
cations.  
fill burn  
1958, F  
craniop  
4-year  
method

## EXPERIENCES WITH ACRYLIC PLASTIC FOR CRANIOPLASTIES

PAUL J. ROSS, M.D., AND FRANKLIN JELSMAN, M.D.

Louisville, Kentucky

The one-stage method of cranioplasty, using acrylic plastic material has been employed by us since 1954. It is the purpose of this paper to present our experiences with 30 consecutive cases. We would also like to present our ideas regarding the advantages of this type of cranioplasty, and the ease with which it may be done.

Plastics have been used successfully by dentists for a number of years. Orthopedic surgeons also have utilized plastics for the reconstruction of arthritic joints; however, they did not prove satisfactory.<sup>4</sup>

Reitz reports that Zander, in 1940, was the first to perform a cranioplasty with acrylic plastic.<sup>5</sup> Since that time, there have been several pioneers in the use of plastics for the repair of cranial defects.<sup>1, 7, 10, 14</sup> In 1948, Oliver and Blaine reported the successful use of a plastic which had been shaped and allowed to harden *in situ*.<sup>7</sup> There were 3 successful cases, which had been followed for 1 year. Spence also described a method of repairing cranial defects with plastics and presented this work at the District of Columbia Scientific Assembly in 1948.<sup>10</sup> Woringer has been one of the chief advocates of this type of cranioplasty. He presented 15 cases in 1951, describing his method in detail.<sup>14</sup> While visiting the Mayo Clinic in 1953, Woringer reported his work using this material. Dodge and Craig adopted this method and reported on their experiences with acrylic cranioplasties in 1953.<sup>1</sup> Their paper was based on animal experiments and repair of 7 cranial defects with plastic. Spence presented a detailed description of his method of repairing cranial defects with a refined methylmethacrylate in 1954, including 5 cases that had been entirely satisfactory for 6 years.<sup>11</sup> Recently, Spence reported that some 50 or more cranioplasty procedures had been done for skull defects over a 10-year period, with very few complications.<sup>10</sup> He had also utilized this material to fill burr holes and trephines in cranial flaps. In 1958, Reitz presented his experiences with 51 cranioplasties, which had been performed over a 4-year period.<sup>8</sup> He had employed the one-stage method for all of these repairs.

The material used for forming these plastic plates is supplied in a kit containing 3 vials of a sterile liquid monomer, and 3 packages of a sterile powdered polymer. It has been suggested by Woringer that the liquid monomer has a bacteriostatic action.<sup>13</sup> The 2 materials should be thoroughly mixed to assure sterilization. By animal experimentation, Blaine and Hoffman, and Dodge and Craig, have shown that there is



FIG. 1. The fibrous dysplasia involving the frontotemporal region is shown in this photograph.

minimal tissue reaction to the plastic plate.<sup>1, 7</sup>

Our method of using plastics for the repair of cranial defects has proven very satisfactory. It is quite similar to the method described by others.<sup>1, 2, 8</sup> The sterile powdered polymer is placed in a small aluminum mixing pan, and the sterile liquid monomer is added. Using a spatula, the two are thoroughly mixed. A period of 7 to 8 minutes is required before the doughy mass is of such consistency that it can be worked satis-



FIG. 2. The extent of bony removal is shown in anteroposterior view

factorily. When the mixture has reached this stage, we have found that pouring some Ringer's solution over the gloved hands and the material, will prevent it from sticking to the gloves. After

a sheet of necessary thickness has been formed by molding with the hands, which we prefer, or by rolling the material on a flat surface, the defect is filled and a form fitting plate of necessary

FIG. 3  
can be o  
ridge.

thickness  
allowed  
ency tha  
contour  
polymer  
During  
tinuously  
mately 7  
excess m  
using a r  
plate; ho  
numerou  
We fix th

There  
plastics  
lying du  
should b  
Adequat  
2 cm. be  
fashion a  
been anc  
bulging  
fashion a  
nature, a  
lumbar n  
necessary



FIG. 3. This illustrates the cosmetic result that can be obtained in reconstructing a supraorbital ridge.

thickness and contour is fashioned. The plate is allowed to harden *in situ* until it is of a consistency that it can be removed without altering the contour of the plate, and before the heat of polymerization will injure contiguous tissues. During this period, the plate is moistened continuously with Ringer's solution. In approximately 7 to 9 minutes, the plate is removed. The excess material and sharp edges are trimmed, using a rongeur and rasp. We do not perforate the plate; however, there are some who think that numerous holes increase stability and fixation.<sup>8</sup> We fix the plate, utilizing braided wire.

There are a few points to observe when using plastics for repair of skull defects. (1) The underlying dura, or exposed brain as the case may be, should be protected by moistened cottonoid. (2) Adequate exposure is necessary for at least 1 to 2 cm. beyond the edges of the defect, in order to fashion a properly fitting plate. (3) If a child has been anesthetized with ether, there may be some bulging at the defect, making it very difficult to fashion a plate. When we have a problem of this nature, a spinal puncture needle is inserted in the lumbar region, so that fluid may be removed if necessary. (4) The plate should be fashioned the

same thickness as the bone in the area which one is working, in order to obliterate any dead space.

(5) A thin flange is allowed to extend around the periphery of the defect, to lend stability and safeguard against any outside pressure forcing the plate inward.

The use of this material to repair cranial defects was first employed by us in July of 1954. From that time through June 1959, we have performed 12 primary and 18 secondary repairs. There were 11 primary repairs after removal of benign bony tumors, or tumorlike lesions,<sup>6</sup> and 1 primary repair after trauma.

The primary repairs were done after the removal of various types of lesions. There were 2 cases of eosinophilic granuloma of the skull, 4 cases of fibrous dysplasia, and 1 each of the following: epidermoid, hemangioma, traumatic cyst, leptomeningeal cyst and benign osteoma. The hemangioma and a case of fibrous dysplasia involved the frontotemporal region. Removal of the supraorbital ridge and roof of the orbit was required in both cases to effect a total removal. The case of fibrous dysplasia is illustrated (figs. 1 to 3). The 1 case of primary repair after trauma was to fill a defect secondary to a compound

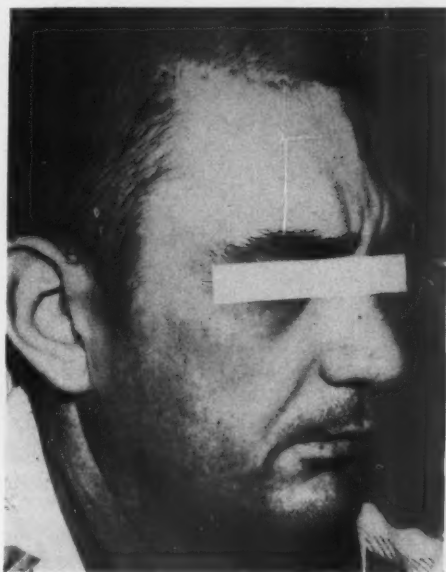


FIG. 4. This photograph shows the defect in the frontal region after surgical debridement of a compound, comminuted, depressed skull fracture.

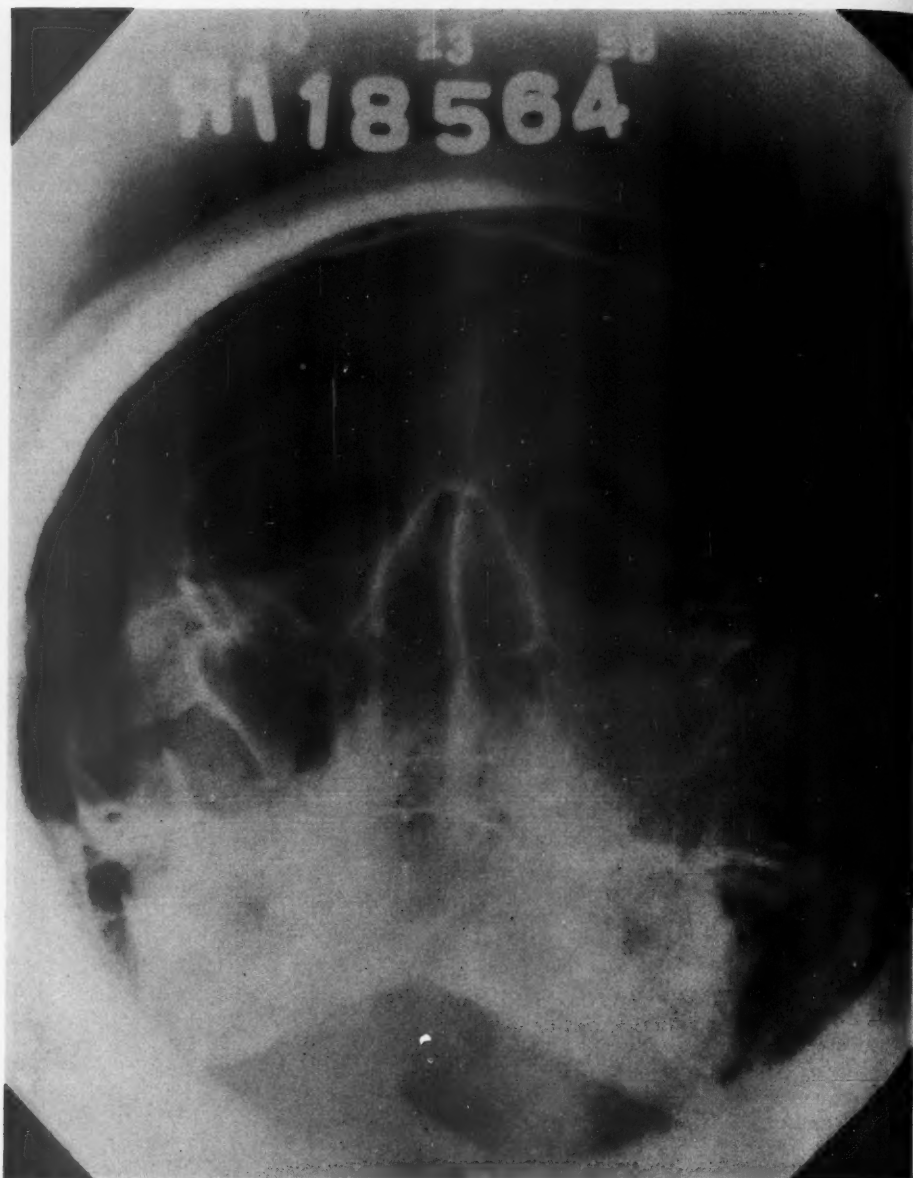


FIG. 5. The defect in the frontal bone, supraorbital ridge and orbital plate, can be seen in this view

depressed skull fracture in the frontal region: the frontal sinus was not involved. However, there are some that routinely use this material for primary repair even when the sinus is involved.<sup>8</sup> Removal of the mucous membrane and plugging

of the sinus with muscle before the plate is fixed in place is advocated in these cases.

Of the 18 secondary repairs, 10 were for defects secondary to trauma and 8 were for repair of surgical defects. The 10 secondary cranioplasties

FIG.  
compl  
days p

after t  
sulting  
There  
region,  
sinus w  
initial  
stripped  
illustra  
after  
skull fr  
depress  
healing  
earlier  
allowe  
formin  
early s  
any co

The  
includ  
tantal  
which  
dural,  
defect  
made  
The 2  
scalp

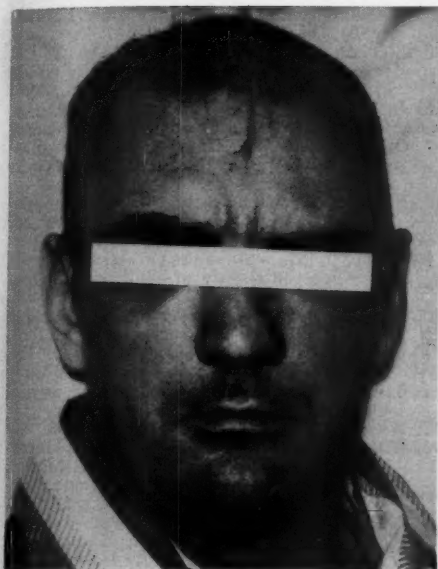


Fig. 6. The cosmetic repair that can be accomplished is shown in this photograph, taken 10 days postoperatively.

after trauma were for the repair of defects resulting from compound depressed skull fractures. There were 6 of these defects in the parietal region, and 4 in the frontal region. The frontal sinus was involved in 2 cases. At the time of the initial surgery, the mucous membrane was stripped from the sinus involved. One case is illustrated that had a secondary repair 3 weeks after debridement of a compound depressed skull fracture (figs. 4 to 6). Recently, in compound depressed fractures, when there has been primary healing, we have been doing a plastic repair earlier than originally. Early in this series, we allowed a period of 3 to 6 months before performing a cranioplasty. Repair has been done as early as 3 weeks after the initial injury, without any complications.

The secondary repairs after surgical defects, included 2 defects that had been repaired by tantalum plates elsewhere. There were 4 defects which had been made in order to remove extradural, or subdural hematomas, and 1 massive defect, measuring 15 cm. by 30 cm., that was made in removing a parasagittal meningioma. The 2 tantalum plates had eroded through the scalp and were infected. These plates were re-

moved, and 3 to 6 months later, plastic plates were fashioned to fill these defects. The plastic plates have been tolerated in both instances.

No plate has been removed as a result of infection or other complications. The cosmetic and functional results have been good in 29 cases, and fair in 1 case. The case in which the result is considered fair was a child that had been anesthetized with ether. Bulging of the underlying structures made it very difficult to fashion a plate. This experience prompted us to employ drainage of spinal fluid by the lumbar root, when we anticipate this problem. This does not mean to imply that this method is used when there is bulging at the defect secondary to increased intracranial pressure. A cranioplasty should not be done when a defect is serving as a decompression.

The rapidity with which a plate can be fashioned is a distinct advantage. Usually, 20 to 25 minutes is required to fashion and secure a plate. It is much easier to attain a good cosmetic result, particularly in those cases involving repair of the supraorbital ridge. Recently one of our colleagues in plastic surgery has utilized this material for reconstruction of the floor of the orbit and nasal bones, with a satisfactory result so far.<sup>12</sup> The plastic plates do not interfere with future x-ray studies. This is particularly helpful in cases of primary tumors of the skull, as the margins of the defect can be seen, and recurrence of the tumor detected. Air studies, angiograms and electroencephalograms can be interpreted without difficulty.

Dead space is obliterated when a properly fitting plate has been fashioned, except in those cases where there has been an extensive internal decompression, secondary to removal of damaged brain, or a tumor. Obliterating dead space affords a more physiologic repair. These plates afford good protection. A number of these plates have been placed in young men who are quite active in sports. There has been no instance in which the plates have not functioned satisfactorily.

We are unaware of any distinct disadvantages associated with the use of acrylic plastic material for the repair of skull defects. To our knowledge, there is no evidence that this material is carcinogenic. We are aware of one report of fracture of a plastic plate as a result of trauma.<sup>5</sup> There is another report of a patient who sustained heavy direct trauma, without fracture.<sup>8</sup>

## SUMMARY

1. A brief resume of the development of acrylic plastic plates is given.

2. A method of one-stage repair of skull defects with acrylic plastics is presented.

3. Thirty consecutive plastic repairs have been done from July 1954 through June 1959, without any complications. Twelve of these were primary repairs; eighteen were secondary repairs.

4. The advantages and disadvantages of acrylic plastic plates are discussed.

Dr. Paul J. Ross

Heyburn Building

Louisville 2, Kentucky

## REFERENCES

1. DODGE, H. W., JR., AND CRAIG, W. M.: Acrylic cranioplasty: a newer rapid method for the repair of cranial defects; preliminary report. *Proc. Staff Meet. Mayo Clin.*, 28: 256, 1953.
2. GURDJIAN, E. S., AND WEBSTER, J. E.: *Head Injuries*. Little, Brown & Co., Boston, 1958.
3. GURDJIAN, E. S., WEBSTER, J. E., AND BROWN, J. C.: Impression technique for reconstruction of large skull defects. *Surgery*, 14: 876, 1943.
4. HARMON, P. H. Methylacrylate resins in surgery. With special reference to the reconstruction of arthritic joints. *Mod. Plastics*, 21: 56; 114, 1942.
5. JACKSON, I. J., AND HOFFMANN, G. T. Depressed comminuted fracture of a plastic cranioplasty. *J. Neurosurg.*, 13: 116, 1956.
6. JELSMA, F. *Primary Tumors of the Calvaria*. Charles C Thomas, Springfield, Ill., in press.
7. OLIVER, L. C., AND BLAINE, G.: A new one-stage method of cranioplasty with acrylic plastic. *M. Press*, 220: 167, 1948.
8. REITZ, K. A. The one-stage method of cranioplasty with acrylic plastic. *J. Neurosurg.*, 15: 176, 1958.
9. SMALL, J. M., AND GRAHAM, M. P. Acrylic resin for the closure of skull defects: preliminary report. *Brit. J. Surg.*, 33: 106, 1945.
10. SPENCE, W. T. Ten years experience using form-fitting plastic for cranioplasty. *Bull. Georgetown Univ. Med. Center*, 10: 154, 1957.
11. SPENCE, W. T. Form-fitting plastic cranioplasty. *J. Neurosurg.*, 11: 219, 1954.
12. WEETER, J. C. Personal communication.
13. WORINGER, E. Nouvelle technique ultra-rapide pour la fermeture de brèches crâniennes avec une résine acrylique autopolymérisable. *Acta chir. belg.*, 51: 655, 1952.
14. WORINGER, E., SCHWIEG, B., BROGLY, G., AND SCHNEIDER, J. Nouvelle technique ultra-rapide pour la réfection de brèches osseuses crâniennes à la résine acrylique. Avantages de la résine acrylique sur le tantale. *Rev. neurol.*, 85: 527, 1951.

The  
arteria  
not on  
Yet de  
forms f  
empha  
upper  
lesions  
treated  
reache  
results

The  
attenti  
affect  
followi

I. T  
A  
B  
II. O  
A

III. E

IV. T

A

B

C

D

E

V. T

VI. C

a

A

B

C

D

E

F

G

H

I

J

K

L

M

N

O

P

Q

R

S

T

U

V

W

## ARTERIAL LESIONS OF THE UPPER EXTREMITY: A CLINICAL REVIEW WITH ILLUSTRATIVE CASES\*

JAMES D. HARDY, M.D., F.A.C.S.†

Jackson, Mississippi

The gratifying progress achieved in the field of arterial surgery over the past decade has involved not only pathophysiology but also management. Yet despite the intense interest which improved forms of treatment have created, relatively little emphasis has been accorded the arteries of the upper extremity (fig. 1). Surgically remediable lesions often remain undiagnosed and thus untreated until a stage of the disease has been reached which precludes optimal therapeutic results.

The purpose of this presentation is to focus attention upon certain conditions which may affect the arteries of the upper extremity. The following will be considered:

- I. *Thoracic (costocervical) outlet syndromes*
  - A. Scalenus anticus
  - B. Cervical rib
- II. *Occlusive disease of major arterial divisions*
  - A. Subclavian, axillary, or brachial artery thrombosis (chronic)
- III. *Embolism*
- IV. *Traumatic lesions of the larger arteries*
  - A. Laceration or division
  - B. False aneurysm (pulsating hematoma)
  - C. Compression and/or spasm
  - D. Thrombosis (acute)
  - E. Arteriovenous fistula
- V. *True aneurysm*
- VI. *Conditions involving the smaller arteries and arterioles*
  - A. Raynaud's disease
  - B. Thromboangiitis obliterans (a distinct disease entity?)
  - C. Vibrating tool (percussion) syndromes
  - D. Reflex neurovascular dystrophy
  - E. Obliterative atherosclerotic disease
  - F. Miscellaneous still less well understood conditions such as the syndrome of the middle-aged woman and scleroderma

\* Aided in part by the United States Public Health Service Grant No. H-2806 (C2).

† Professor and Chairman, Department of Surgery, and Surgeon-in-Chief to the University Hospital, University of Mississippi Medical Center, Jackson, Mississippi.

### THORACIC OUTLET (COSTOCERVICAL) SYNDROMES

Anatomic derangements at the thoracic outlet may result in compression of the subclavian artery or the brachial plexus, or both.<sup>1, 4, 14, 15, 26, 29</sup> A number of different more or less distinct conditions have been described, but the two most prominent ones are the scalenus anticus and the cervical rib syndromes. Actually, these two conditions may overlap. A taut scalenus anticus muscle which compresses the subclavian artery may or may not be associated with a cervical rib; or, the compression may be produced entirely by a cervical rib with no assistance from the scalenus anticus muscle. Nevertheless, it serves clarity to consider the scalenus anticus syndrome and the cervical rib syndrome as separate entities, as long as it is realized that in some patients elements of both may be present. Prolonged hyperabduction of the arm may in itself result in vascular changes.<sup>7, 63</sup>

#### *Scalenus Anticus Syndrome*<sup>12, 44</sup>

*Anatomic considerations.*<sup>26, 56</sup> The subclavian artery emerges from the thorax by passing between the scalenus anticus and the scalenus medius muscles (fig. 2). The fact that it lies caudad to, but immediately along the lower border of, the brachial plexus is of considerable practical importance. There exist at times firm fibrous bands which may sharply define the posterior border of the scalenus anticus or the anterior border of the scalenus medius muscle. In one of our cases it appeared that such a band might have been in part responsible for the compression of the subclavian artery when certain postures of the thorax or of the arm on the involved side were assumed.

*Pathophysiology.* Precisely how or why the scalenus anticus muscle produces subclavian artery compression in one person and not in another is not entirely clear. Some workers have maintained that this muscle has a broader insertion along the first rib in patients so afflicted,

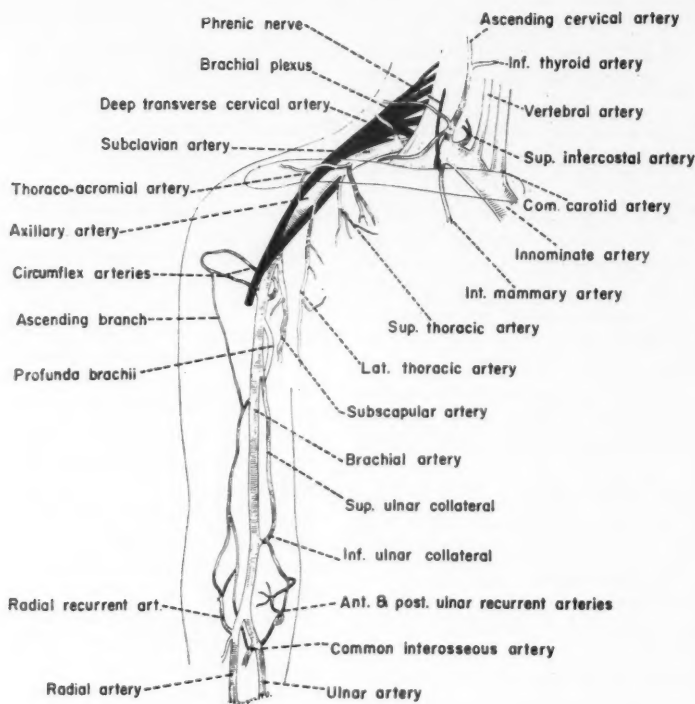


FIG. 1. Arterial supply to the arm. Every effort should be made to restore a pulsatile flow through arteries occluded, compressed, or otherwise diseased or injured. Primary anastomosis of severed radial and ulnar arteries has been successfully performed.

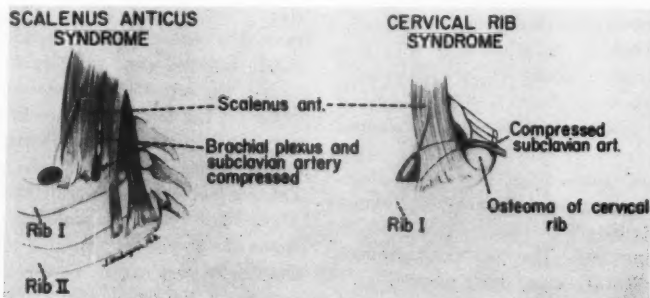


FIG. 2. Anatomic relationships in scalenus anticus and cervical rib syndromes

whereas others have claimed that the pathology consists of an increased firmness and hypertrophy of the muscle. In a case to be described below, the patient had a long neck and sloping shoulders. The compression of the artery was produced by turning the head sharply to the involved side (fig. 3) or by abduction of the arm on that side,

or both. A similar but less marked reduction in the blood pressure and the quality of the radial pulse were effected by turning the head sharply to the unaffected side. At operation, with the exposed subclavian artery and scalenus anticus muscle in full view, it was seen that these maneuvers did in fact cause the scalenus anticus muscle to

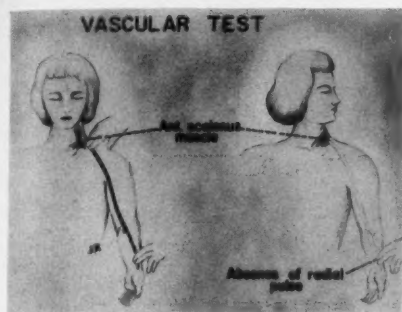


FIG. 3. Adson test for costocervical compression of the subclavian artery. In the scalenus anticus syndrome, with or without cervical rib, the radial pulse may be obliterated by turning the head sharply toward the affected side. The effect may be increased, or produced independently, by hyperabduction of the arm on the involved side.

enroach upon the artery, resulting in markedly reduced pulsations distal to the point of compression. The muscle itself did not appear abnormal or otherwise remarkable. The scalenus anticus syndrome is more common in women than in men.

**Diagnosis.** It is usually the *symptoms* relative to ischemia of the arm or hand or to neural sequelae which lead the physician to suspect the presence of scalenus anticus compression. Frequently the patient will complain of paresthesia, tingling, pain, fatigue or numbness in the extremity when it is elevated above the head, sharply abducted, or when she sleeps in certain positions. There may be episodes of cyanosis of the fingers, at times due to secondary Raynaud's phenomenon and not necessarily due to marked subclavian artery compression. Pain may involve not only the extremity but also the supraclavicular fossa and the side of the neck as well. **Physical examination** may reveal equal pulses and blood pressure measurements in the two upper extremities when the patient is supine with her arms at her sides. However, even in this position a bruit may at times be heard in the supraclavicular fossa. In marked contrast to the arm-at-side position, when the affected arm is elevated above the head and abducted<sup>7, 8</sup> the pulse and blood pressure in this arm may be obliterated—especially if the head is simultaneously turned toward the affected side (fig. 3). Turning the head to the opposite side may have an almost equal effect. These same maneuvers produce far

less or no effect in the unaffected or asymptomatic arm.

At the same time that the elevation of the arm produces a reduction in the volume of the radial pulse, a bruit may be heard and a thrill felt over the supraclavicular fossa. This may be rendered even more audible by having the patient take a deep breath. If the scalenus anticus compression has resulted in poststenotic dilation ("aneurysm" formation) of the subclavian artery—which is less common here than when a cervical rib is present—a pulsating mass may be detected.

The *differential diagnosis* of scalenus anticus pain includes consideration of the various conditions listed in table 1. Thrombosis and embolism are excluded by the presence of a normal radial pulse and blood pressure when the arm is at the patient's side. Nevertheless, it will be seen below that thrombosis of the subclavian artery and distal embolism are not uncommon complications of the cervical rib syndrome, where there may also be a scalenus anticus component. Finally, the mere fact that the Adson vascular test (fig. 3) may almost obliterate the pulse and blood pressure in a given individual does not constitute a genuine scalenus anticus syndrome

TABLE 1

*Some causes of chronic shoulder, arm, or hand pain*

1. Vascular lesions
  - a. Scalenus anticus syndrome
  - b. Cervical rib syndrome
  - c. Arterial thrombosis
  - d. Aneurysm
  - e. Raynaud's disease
  - f. Thromboangiitis obliterans
  - g. Vibrating tool (percussion) syndromes
  - h. Reflex neurovascular dystrophy
  - i. Obliterative atherosclerotic disease of terminal arteries
2. Nerve lesions
  - a. Cervical disc (herniated)
  - b. Spinal cord tumor
  - c. Superior sulcus tumor
  - d. Neoplasm of brachial plexus (as in neurofibromatosis)
  - e. Peripheral neuritis
  - f. Traumatic injury to brachial plexus
3. Miscellaneous
  - a. Cervical osteoarthritis
  - b. Bursitis
  - c. Referred pain from the diaphragm, heart or biliary tract

### APPROACH TO SUPRACLAVICULAR SPACE

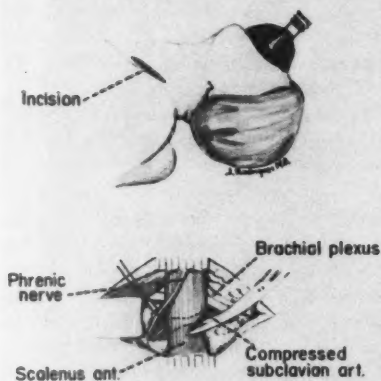


Fig. 4. A short incision parallel to the clavicle in the supraclavicular fossa affords satisfactory exposure of the scalenus anticus muscle, cervical rib, subclavian artery and brachial plexus.

in the absence of symptoms. In any group of medical students, internes and residents making ward rounds, a completely asymptomatic person may be detected in whom the Adson test is positive for subclavian artery compression. Actual ischemic necrosis of the fingers is rarely caused by scalenus anticus compression in the absence of a cervical rib.

**Treatment.** Physical therapy, proper instruction and reassurance will suffice to relieve mild symptoms referable to the scalenus anticus syndrome in many instances. In some patients, however, the condition imposes genuine physical limitations which require surgical correction. This consists of exposure and division of the offending scalenus anticus muscle near its insertion on the 1st thoracic rib (fig. 4), as described in the case now to be presented.

#### Case 1. Scalenus Anticus Syndrome without Cervical Rib

D. K., an 18-year-old unmarried white girl, was admitted to the University Hospital on May 26, 1959. She had been well until approximately 1 month previously when she had begun to have pain in the left shoulder beginning in the supraclavicular area and radiating up the lateral aspect of the neck, ending just behind the left ear. She also had pain radiating down the left arm and involving the fingers. The pain was not always present, but usually began suddenly and was

aching in nature. Associated with the pain, it had been noted that the left arm became numb and that the nail beds were quite blue. She had also discovered at one time a swelling in the left supraclavicular area for which she had consulted a local physician who detected a bruit. Over the past 4 weeks she had experienced decreasing strength in the left arm, which was particularly apparent when she tried to use both arms simultaneously in hanging clothes above her head on a clothes line; the left arm quickly tired and had to be lowered. She stated that recently she had developed a left temporal headache when the pain radiated to the arm, associated with throbbing and a feeling of soreness. The periods of pain and of weakness of the arm occurred together. There were no symptoms to suggest that brachial plexus compression might have resulted in nerve injury *per se*.

Physical examination revealed a girl in excellent general health who had a long neck and sloping shoulders. With both arms at her sides, the blood pressures in the two arms were normal and equal. However, when the left arm was raised over the head and rotated outward the blood pressure and radial pulse disappeared entirely, while remaining normal in the right arm. The same obliteration of blood pressure and pulse in the left arm was obtained by the turning of the head sharply to the left while at the same time asking the patient to inhale deeply. No thrill was palpable in the supraclavicular space, but a bruit could be heard on auscultation.

The diagnostic impression was that of "pure" scalenus anticus syndrome, since roentgenograms had disclosed no cervical rib.

At operation on May 28, 1959, the supraclavicular space was explored by means of a short transverse incision (fig. 4). The elements of the brachial plexus were exposed and gently retracted laterally, and the artery was freed up to its point of emergence between the anterior and medial scalene muscles. The subclavian artery distal to the scalenus muscles was definitely subnormal in size. At this point the pulsations in the subclavian artery were examined while the anesthesiologist turned the patient's head sharply, first to the left and then to the right. It was noted that the pulsations in the left subclavian artery were almost obliterated by turning the head sharply to the left, although there was a definite reduction in the volume of flow to this vessel when the head was turned to the right also. Next the phrenic nerve was identified and retracted medially. The scalenus anticus muscle was then severed at a point just above its tendinous insertion (fig. 4). Once the scalenus anticus muscle had been divided anterior to the artery it was apparent that there

remained a fibrous band just posterior to the artery, and it also was divided. After this the finger was used to examine the course of the artery as it entered the thorax and to make certain that no other points of compression existed. There were none, and it was observed that the diameter of the subclavian artery had already increased. The wound was irrigated with saline solution and then closed in layers with catgut in the subcutaneous structures and vertical mattress sutures of silk in the skin.

Postoperatively the Adson vascular test was no longer positive for evidence of compression of the left subclavian artery, and the radial pulse and blood pressure in that arm were equal to those in the right arm in all positions. All symptoms were abolished.

*Comment.* This patient presented a classic scalenus anticus syndrome with intermittent Raynaud's (vasospastic) phenomena. The episodes of ischemia were at times associated with a temporal headache, a finding described in the literature as being a symptom of a cervical rib.<sup>27</sup>

#### *Cervical Rib Syndrome*

*Anatomic considerations.*<sup>26, 58</sup> Normally the subclavian artery and the brachial plexus emerge between the scalenus anticus and scalenus medius muscles just above the first thoracic rib (fig. 2). However, when a cervical rib is present, particularly if it is a complete one articulating with the 1st thoracic rib, the brachial plexus often comes off one segment higher up, and neural compression is not a prominent feature.<sup>49</sup> The subclavian artery, though, may be compressed as it crosses over an enlargement or osteoma at the distal end of the cervical rib (fig. 2). In contrast, when an incomplete cervical rib is short and pointed, a fibrous band may connect the cervical rib tip and the 1st thoracic rib. Here compression of the nerve roots may result in a clinical picture of segmental sensory loss with wasting of the small muscles of the hand, but there is less likelihood that the subclavian artery will be encroached upon.

*Pathophysiology.* It has been seen that the cervical rib may be associated with predominantly neural symptoms,<sup>41</sup> predominantly vascular symptoms, or with no symptoms. The pathologic changes are of course dependent upon the structures compressed. Women are more commonly afflicted than men.

Changes secondary to brachial plexus compression may be either sensory or motor. The

most common and characteristic symptoms are pain and paresthesia in the involved extremity.<sup>5</sup> Later the muscular atrophy,<sup>62</sup> motor weakness and disturbances in sensation may appear. Even when the subclavian artery is not compressed, nerve lesions may give rise to Raynaud's phenomenon with intermittent reflex spastic ischemia of the fingers, or to reflex neurovascular dystrophy suggestive of sympathetic nerve dysfunction as seen in certain types of shoulder-hand syndrome.

*Subclavian artery compression* by a cervical rib may give rise to symptoms of ischemia quite similar to those described under the discussion of the scalenus anticus syndrome. In fact, it is frequently difficult to determine just how much of the symptomatology is due to the scalenus anticus muscle and how much is due to the cervical rib, when a cervical rib is present.

*Vascular complications of cervical rib.*<sup>43, 49, 50, 59</sup> Encroachment upon the subclavian artery may result in one or more of the following, several of which have been alluded to previously: (1) ischemia of the extremity of varying degree, at times with associated Raynaud's phenomenon; (2) poststenotic dilation or aneurysm formation;<sup>34</sup> (3) thrombosis,<sup>47</sup> perhaps in the dilated portion of the artery distal to the point of compression; (4) embolization<sup>47</sup> of parts of thrombus to more distal arteries of the arm; (5) thinning of the wall of the artery at the point of compression, with risk of rupture spontaneously or at the time of surgical intervention.

Whereas the simple scalenus anticus syndrome rarely produces necrosis of a finger tip, all degrees of ischemia may be observed in association with a cervical rib. The milder forms may consist of only slight numbness and tingling of the fingers and forearm, sometimes aggravated when the arm or head is placed in certain positions (fig. 3). At the other extreme, even gangrene of the fingers may result from cervical rib compression, particularly if thrombosis of the vessel is followed by distal embolization. Shenkin<sup>53</sup> reported 3 patients with cervical rib who had sustained subclavian artery thrombosis, all of whom had had mild symptoms for several years preceding this complication. The diagnosis of cervical rib syndrome had been made early, but conservative management had been advised. However, since the thromboses had occurred without a premonitory increase in symptoms, the author advised that

early surgery be seriously considered for a cervical rib causing neurovascular symptoms.

**Diagnosis.** The diagnosis of cervical rib syndrome is not difficult when appropriate symptoms exist and a cervical rib is demonstrated on roentgenogram. Clinical evidence of neural or vascular compression may be augmented by use of the hyperabduction vascular test (fig. 3). Pain in the hand and forearm may be increased by exercise (intermittent claudication) and decreased by resting the extremity. There may be a fullness and a prominent pulsation in the supraclavicular fossa, caused by anterior displacement of the subclavian artery by the cervical rib or by aneurysm formation. The enlarged distal end of a complete cervical rib may be easily palpable, as it was in the case to be described below. A thrill and a bruit may be present, and swelling and coldness of the extremity or fingers may be observed. There may have been intermittent attacks of coldness affecting the hand and paresthesia may be permanent in the fingers with numbness of the fingertips. Shallow indolent ulcers of the fingers by the side of the nail or over the finger pulp may develop, but actual gangrene of the fingers is relatively rare. Should there be a sudden increase in the severity of the symptoms, the possibility of acute thrombosis or embolism should be considered.

**Angiography** may disclose compression, thrombosis, aneurysm formation or evidence of distal embolization.

As in the diagnosis of pure scalenus anticus syndrome, however, one must be careful not to attribute to a demonstrated cervical rib symptoms and physical findings which may be due to some other lesion (tables 1 and 2). A cervical rib may be entirely innocent and frequently is.

**Treatment.** The management of the cervical rib syndrome may be either conservative, with physical therapeutic measures, or surgical. If operation is elected, the technical maneuvers required will depend upon the pathology found. The cervical rib, or at least its more distal portion, should be excised, along with any fibrous band extending from the cervical rib to the 1st thoracic rib. If this entirely liberates the brachial plexus and subclavian artery, division of the scalenus anticus muscle may not be required. Should ischemic type pain have existed preoperatively, perhaps associated with causalgia or neurovascular dystrophy, cervical sympathectomy may be indicated. When dividing the

TABLE 2

*Some arterial lesions which may cause finger necrosis*

1. Scalenus anticus syndrome
2. Cervical rib
3. Arterial thrombosis (chronic) of subclavian, axillary or brachial artery
4. Emboli
5. Traumatic injury (acute)
6. Raynaud's disease (rarely)
7. Thromboangiitis obliterans
8. Atherosclerosis obliterans
9. Vibrating tool (percussion) injury
10. Scleroderma (a vascular disease?)

scalenus anticus muscle at or near its insertion through a short transverse incision in the supraclavicular fossa, the phrenic nerve must be identified and retracted out of harm's way. Great care should be taken to retract the elements of the brachial plexus gently when excising the cervical rib, for postoperative symptoms not present preoperatively are at times a direct result of retraction of the nerve roots, as it was in the following case.

#### *Case 2. Cervical Rib Syndrome*

S. P., a 15-year-old white school girl, was well until July 1958, when, after an afternoon of diving from a high diving board, she became aware of aching pain in the left supraclavicular area and shoulder. For the first time she and her mother noted a small lump just above the clavicle. Thereafter from time to time the left shoulder pain recurred, associated with soreness in the left supraclavicular fossa and at times along the left arm. The family physician was consulted in November 1958, and the diagnosis of a possible aneurysm or other anomaly of the left subclavian artery was made. She was referred to the writer on December 30, 1958, and the previous finding of a mass (2 by 2 cm.) in the supraclavicular area was confirmed. There was a slight thrill over the mass, and a soft bruit was heard. The mass itself was firm and only slightly movable, if at all, and was nontender.

A roentgenogram disclosed that the patient had bilateral cervical ribs, and that the mass probably represented an osteoma or enlargement of the distal end of the left cervical rib, the side on which the patient had symptoms. The left cervical rib was a complete one and it appeared to be fused with the 1st thoracic rib.

Changes in position of the extremity had little effect upon the blood pressure, the value being

110/60 in the right arm and 112/60 in the left arm. A diagnosis of left cervical rib syndrome was made and the patient was admitted to the University Hospital on January 8, 1959.

At operation on January 9, 1959, performed under general anesthesia, the left supraclavicular space was explored through a transverse incision similar to that shown in figure 4. As the dissection was developed to include the areolar tissue and muscle overlying the brachial plexus, a rather small artery was identified and indifferently preserved, but this small vessel subsequently proved to be in fact the left subclavian artery just distal to its passage over the mass which was shortly demonstrated to be the enlarged distal end of the left cervical rib (fig. 2.) The left subclavian artery was sharply compressed by this enlargement. The distal end of the cervical rib lay behind the 1st thoracic rib, and its enlarged distal end was joined to the latter structure by firm and dense fibrous tissue. After the subclavian artery and the structures of the brachial plexus had been retracted out of harm's way, the distal two-thirds of the left cervical rib were resected, the most difficult maneuver being the separation of the enlarged distal end from its firm fibrous union with the undersurface of the left thoracic 1st rib. However, this was accomplished expeditiously and without event. Once this cervical rib had been removed there was a prompt enlargement in the diameter of the left subclavian artery.

Postoperatively this patient complained for some weeks of mild paresthesia in the left hand, particularly along the ulnar surface. The symptoms were unquestionably the result of the changes in and around the brachial plexus adjacent to the site of operation. This occurred despite the fact that the members of the operating team were well aware of the hazard of postoperative neurologic symptoms after operations in the area of the brachial plexus. Fortunately these rather mild neurologic symptoms in the left arm and hand subsided with appropriate physical therapy, and the patient was symptom-free some 9 months later when last seen and discharged from observation. No sensory or motor loss was observed at any time.

*Comment.* This is the second patient that the writer has operated upon who had mild to moderate but fairly prolonged neurologic symptoms along the ulnar surface of the arm and hand after resection of a cervical rib. It is again emphasized that retraction of the elements of the brachial plexus—and, indeed, of any nerve anywhere—should be as gentle and minimal as is compatible with adequate exposure to prevent injury. This case also illustrates a number of considerations

which are involved in the decision regarding whether or not to excise a symptomatic cervical rib. There was no question but that the patient had more discomfort in the left arm and hand following resection of the rib than she had had preoperatively, until the neurologic symptoms had subsided. Nevertheless, she had had a mass associated with a bruit and a thrill, indicating the compression of the left subclavian artery which was demonstrated and corrected at surgery. As noted above, Shenkin<sup>23</sup> has called attention to the fact that patients who are allowed to continue with definite evidence of compression of the subclavian artery by a cervical rib may eventually develop acute thrombosis of the subclavian artery with or without distal embolization. This train of events can result in considerable disability and weakness of the extremity.

Our patient still has the cervical rib on the right side. Since it has caused no symptoms, operation has not been advised. Whether or not surgery will be recommended in the future will depend upon a long-term follow-up. Should evidence of right subclavian artery compression develop, surgery will be given serious consideration. However, whereas there was enlargement of the distal end of the cervical rib on the left, which could be palpated and could be demonstrated on roentgenogram, there is no similar enlargement of the complete cervical rib on the right. Compression of the subclavian artery may never develop.

#### OCCLUSIVE DISEASE OF MAJOR ARTERIES

The atherosclerotic occlusive disease which may affect any artery in the body may also affect the elements of the arterial supply to the upper extremity. Although it was seen above that thrombosis of the subclavian artery with or without embolization may be secondary to compression syndromes such as that caused by a complete cervical rib, the compression element need not be present for atherosclerotic thrombosis of the subclavian, axillary, or brachial artery to occur.

Occlusive disease is usually a slowly developing chronic process, but it may represent acute thrombosis or fresh thrombosis superimposed upon chronic thrombosis. Whereas the thrombosis may initially involve only a small segment of the artery and be amenable to thromboendarterectomy, distal and proximal propagation



FIG. 5. Gangrene of fingertip due to occlusion of axillary artery (see also fig. 6). Thromboangiitis obliterans had been suspected until the almost complete occlusion of the axillary artery had been demonstrated (case 3).

of the thrombus may eventually render corrective surgery impossible. Early diagnosis and prompt treatment are essential if good results are to be achieved in a high percentage of patients.

The patient with occlusion of the axillary artery, for example, may exhibit all the usual symptoms resulting from ischemia, and finger necrosis may be observed (fig. 5). The diagnosis of thromboangiitis obliterans may have been erroneously made previously; or if secondary Raynaud's phenomenon is present, he may have been considered to have Raynaud's disease or some other condition of the smaller distal arteries (table 2). In at least one instance of our acquaintance the patient had had a cervical sympathectomy for what was believed to be Buerger's disease involving the hand before it was demonstrated that he had no radial, brachial, or axillary pulse distal to a given point; above this level the pulsations were normal.

The other symptoms and signs of occlusion of the subclavian, axillary and brachial arteries are those which reflect arterial ischemia anywhere. Naturally all accessible arterial pulses should be recorded as being present or absent in all patients suspected as having vascular disease—and even in patients who as yet have no vascular symptoms.

We have encountered in recent months 5 patients who had evidence of occlusive disease of the subclavian or axillary artery. One of these was an elderly housewife who noted only that her right arm and hand became fatigued when she

did her housework vigorously. It was found that she had a right carotid pulse but absent right subclavian, brachial, and radial pulses. The right arm was cooler than the left, but there was no evidence of impending necrosis of the fingertips. The patient had no symptoms other than the intermittent claudication when she exercised the right extremity. She did not desire surgery and was discharged.

The 2nd patient was a man who had a lowered blood pressure in the left arm as compared with the right. He also had a marked arterial occlusive disease in the lower extremities, and at laparotomy he was found to have a fairly large aneurysm of the abdominal aorta that was completely occluded with old thrombus. This was uneventfully resected, and a Teflon prosthesis inserted. He has not been readmitted for further evaluation of the diminished blood pressure in the left arm, although partial subclavian occlusion is likely. There is no cervical rib, and there is no evidence of a scalenus anticus syndrome. He has not had attacks of dizziness which would suggest basilar artery insufficiency due to occlusion of the left vertebral artery.

The 3rd patient had been treated repeatedly for "Buerger's disease" of the fingers of the left hand. On admission to the University Hospital it was apparent that he had occlusion of the left subclavian artery in its second portion. At surgery (fig. 7) a thrombus was removed which extended from the subclavian artery, through the axillary artery, to the proximal portion of the

brachi  
the br  
helpfu  
consid  
the ex  
lasted  
revers  
exam  
have  
with t  
of ma  
earlier  
is awa  
case o  
be for

Firs  
was fi  
April  
eral a  
teries  
and c  
mitter  
consid  
cessat  
pulse  
pulses  
leg wa  
eviden  
Fur  
and a  
logic



FIG. 6. Arteriogram demonstrating occlusion of axillary artery (case 3).

brachial artery. Here retrograde flushing through the brachial artery was found to be especially helpful in the removal of the more recent clot. A considerable increase in blood flow was afforded the extremity, but the ischemic condition, having lasted for many months, was not satisfactorily reversed. This patient represented an excellent example of a patient in whom it probably would have been possible to achieve a good result with thromboendarterectomy, had the diagnosis of major arterial occlusive disease been made earlier and operation performed promptly. Case 4 is awaiting admission to the hospital. The 5th case of atherosclerotic occlusive disease will now be formally described.

### Case 3. Segmental Thrombotic Occlusive Disease of Left Axillary Artery

**First admission.** E. R., a 73-year-old white man, was first admitted to the University Hospital on April 2, 1956, with a tentative diagnosis of peripheral arterial occlusive disease involving the arteries to the legs. He gave a history of coldness and cyanosis of the feet associated with intermittent claudication on the left that had been considerably relieved by Priscoline therapy and cessation of smoking. Although the left femoral pulse was weak and the left popliteal and foot pulses were absent, and although the left lower leg was cooler than the right, there was little evidence of tropic changes in the foot.

Further evaluation disclosed an unsteady gait and a moderate left facial weakness. The neurologic consultant rendered the opinion that the

### INCISIONS FOR EXPOSURE OF ARTERIES TO UPPER EXTREMITY

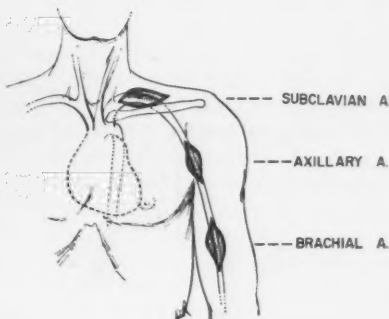


FIG. 7. For exposure of the left subclavian artery at its origin a sternal splitting incision is useful. The axillary artery can usually be approached through an incision in the deltoid-pectoral crease, without division of the tendinous portion of the pectoralis major muscle at its insertion on the humerus.

patient probably had had a mild recent cerebrovascular accident and that major lower aortic surgery was contraindicated. (At that time the members of the surgical staff were not yet aggressively alert to the possibilities of the demonstration and correction of occlusive lesions of the internal carotid arteries and the vertebral arteries.) Thus after evaluation the patient was discharged, since the arterial disease of his left leg was not incapacitating.

**Second admission.** The patient was next seen at the University Hospital on January 4, 1959. During the almost 3 years since his first admission he had done rather well and had had no significant increase in the moderate ischemia of the left leg. However, 3 weeks before, he had had a sudden onset of severe pain in the left hand while pouring gasoline into the tank of a truck in cold weather. This pain had subsided when he warmed his left hand, and he had had no further difficulty until 2 days before, when he noticed the onset of coldness and severe pain in all fingers of the left hand. At that time marked cyanosis was noted in these fingers, and there was an inability to accomplish fine movements. He stated that the intermittent claudication in the left thigh was still present. The left radial pulse was weak and intermittently absent.

He was treated with multiple left stellate ganglion blocks for several days, and as this had afforded considerable relief of the symptoms in the left hand he was discharged. Nevertheless,

## THROMBOENDARTERECTOMY

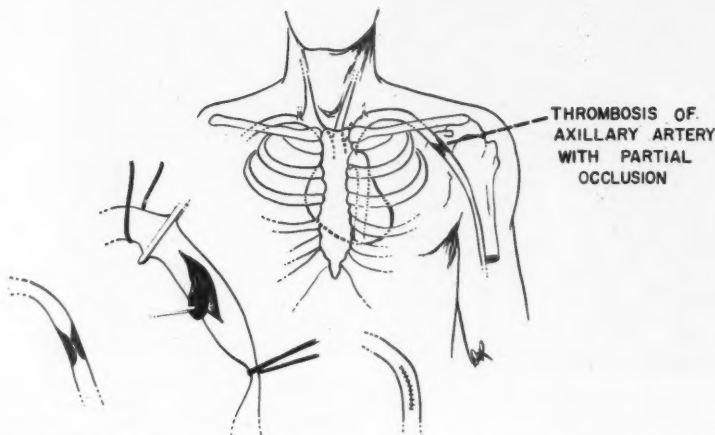


FIG. 8. Removal of a chronic thrombus by endarterectomy can be highly successful if performed before extensive proximal and distal clot propagation has occurred. Early diagnosis increases substantially the incidence of successful surgery.

relief was short-lived, and he had to be hospitalized again.

*Third admission.* After entirely too much preliminary conservative management, during which time the tip of the little finger of the left hand was allowed to become gangrenous (fig. 5), the stellate ganglion blocks were discontinued, and the patient was readmitted after peripherovascular consultation. The vascular consultant promptly made a diagnosis of organic block of the axillary artery, rather than distal obliterative atherosclerosis of the smaller vessels of the fingers. This diagnosis was confirmed with an arteriogram (fig. 6). At operation on February 9, 1959, the left subclavian artery was exposed (fig. 7) and a tape passed around it, as was the axillary artery distal to the point of occlusion (fig. 8). Several other branches were temporarily controlled with loops of silk. A longitudinal incision was then made directly over the thrombus, which was approximately 2 cm. in length, and a thromboendarterectomy was performed quite easily. Before endarterectomy there had been no pulse in the exposed artery distal to the site of the thrombosis, but afterward there were excellent axillary, brachial and radial pulses.

*Postoperatively* the hand pain was largely abolished, although some pain persisted in the necrotic tip of the 5th finger. The coldness of the left hand did not disappear at once but gradually receded, and the patient was discharged from the hospital on February 19, 1959, much improved. Thereafter, he continued to be observed in the follow-up clinic. By August 1959, the left hand was essentially

asymptomatic, and the previously necrotic tip of the left 5th finger had healed almost entirely.

He has continued to have some intermittent claudication in the left leg, but thus far neither definitive surgery for this nor carotid and vertebral arteriograms have been recommended. Both common carotid pulses are present.

*Comment.* This patient illustrates many features of the patient who has slowly developing obliterative atherosclerotic disease of various arteries. He had first been seen at the University Hospital in 1956 for inadequate blood flow to the left leg, characterized by the usual signs of ischemia and intermittent claudication. Nevertheless, conservative measures—consisting chiefly of limited exercise, Priscoline, and abstinence from smoking—afforded considerable improvement in the leg symptoms and no operative procedure was ever performed for these complaints. He had also had, it will be recalled, evidence of cerebral ischemia as reflected in a mild stroke. Finally, he was followed in our own out-patient clinic for some time before it was appreciated by a person especially trained in the diagnosis of arterial diseases that he actually had an organic occlusion of his axillary artery rather than atherosclerosis obliterans of the smaller vessels of the hand and fingers. In defense of those who treated him conservatively for such a length of time that the tip of one finger actually

became weakly possible level v lished interm diminu terial pressu This which more of ath any a toms throu would toes v but it possi major thoug

Th the m throm grafts theses passin upper result

Em artery which in the ventr or ve from a pat and t that farcti the t wall deriv Em arter 330 Hain

became gangrenous, the left radial pulse was weakly present from time to time, and thus the possibility of occlusion of the artery above this level was not fully appreciated. It is an established fact that weak arterial pulsations may be *intermittently* present when the volume of flow is diminished by partial proximal occlusion. Arterial spasm and variations in systemic blood pressure may play a role in such pulse changes.

This patient's case also spanned a period during which the staff of our hospital were becoming more and more aware of the widespread nature of atherosclerosis, and the fact that virtually any and all organs may be the source of symptoms due to inadequate arterial blood flow through their supplying vessels. Almost no one would now diagnose "Buerger's disease" of the toes without first palpating the femoral arteries, but it is only in the last year or so that the possibility of atherosclerotic occlusion of the major arteries of the arm has been promptly thought of by most examining physicians.

#### *Concluding Comment on Management of Occlusive Disease*

Thus far we have treated occlusive disease of the major arteries of the upper extremities by thromboendarterectomy only. However, homografts, venous autografts, and synthetic prostheses have been used after resection or for bypassing occlusive lesions of the arteries of the upper extremity, with reasonably satisfactory results.

#### EMBOLISM

Embolism may produce sudden occlusion of an artery. The heart is the usual source of emboli, which are most often derived from the atrium in the presence of atrial fibrillation or from the ventricle in the presence of myocardial infarction or ventricular aneurysm. Other emboli may arise from vegetations on the heart valves, but when a patient has evidence of acute arterial embolism and the heart is not fibrillating, the chances are that he has recently suffered a myocardial infarction whether or not this was appreciated at the time. Of course, partial thrombosis in the wall of an artery may give rise to distal emboli derived from this thrombus.

Emboli from the heart do not often lodge in arteries of the upper extremity. In a review of 330 instances of peripheral arterial embolism, Haimovici<sup>10</sup> found that 38 per cent occurred in

the femoral arteries, 14 per cent in the popliteal arteries, 14 per cent in the common iliac arteries, 9 per cent at the bifurcation of the aorta, and 9 per cent in the brachial arteries. The remainder were in miscellaneous arteries elsewhere. The important fact to be noted is that only about 9 per cent of arterial emboli from the heart become lodged in the arteries to the upper extremities.

*Clinical findings.* In general the symptoms and physical findings of acute arterial embolism are similar to those caused by acute arterial ischemia under any circumstances. There will usually be ischemic pain in the extremity distal to the site of occlusion, coldness, pallor, numbness, diminished motor power may follow, and eventually gangrene may develop.

Surprisingly, in some cases of acute arterial occlusion pain may not develop for many hours and may actually never be a prominent symptom. It is not uncommon in such circumstances for the attending physician to suspect that a nerve lesion is responsible for the changes in motor power and sensation in the extremity. Even in those cases in which the initial symptoms do not include significant pain, however, the pallor and coldness of the extremity should always suggest the possibility of arterial disease in contradistinction to a nerve lesion. The suspicion of embolism is further strengthened by the finding of atrial fibrillation or electrocardiographic evidence of a recent myocardial infarction.

*Treatment and prognosis.* Whereas an embolus to the bifurcation of the common femoral artery might well result in sufficient ischemia to produce gangrene of the toes or foot, particularly if severe arteriospasm were associated with the abrupt occlusion of the artery by the embolus, emboli to the upper extremity do not often result in gangrene of the extremity. The collateral arterial blood supply to the upper extremity is extensive, and thus conservative measures will usually suffice to prevent necrosis. For this reason the tendency in the past has been to employ conservative measures almost exclusively in the presence of arterial embolism to the arteries of the upper extremity. However, such emboli may result in a considerable degree of ischemia of the extremity, which can be seriously disabling in an individual who must use his arm for making his living. Thus in the light of present day knowledge regarding the exposure and suture of most accessible arteries of the body, we would now

operate upon the patient with acute embolism to any artery of the arm which was above the level of the brachial bifurcation, and embolectomy would be performed. If the embolus could not be removed, perhaps because of thrombotic propagation into the ulnar and the radial arteries, the conservative measures of repeated stellate ganglion block, anticoagulants, vasodilator drugs, and even cervical sympathectomy would be employed.

#### TRAUMATIC LESIONS OF THE LARGER ARTERIES

*Pathologic conditions.*<sup>18</sup> The subclavian, axillary, brachial, radial and ulnar arteries may be injured in various ways, although blunt force and stab and gunshot wounds are particularly common etiologic factors. The vessel may be lacerated or completely severed. A false or a true aneurysm may develop, as may an arteriovenous fistula. Compression or spasm may be severe,<sup>28</sup> and intramural or intraluminal thrombosis may produce complete occlusion.

*Diagnosis.* In general the injury is detected through the history which suggests possible arterial injury, physical examination, an arteriogram where indicated, and surgical exploration in selected cases. Sympathetic nerve block may aid in differentiating arterial spasm from arterial interruption or occlusion.

#### Laceration or Division

In our experience laceration or division of an artery of the extremity has most often been due to knife or gunshot wounds, but in one young child the brachial artery had been severed when she fell upon a broken Coca-Cola bottle. This type of arterial injury is usually apparent because of the loss of blood to the exterior, with or without hematoma. If the artery is incompletely divided, hemorrhage may be continuing and may be exsanguinating. In contrast, if the artery is completely divided, as it was in the child injured on the glass bottle, the ends of the artery may retract and hemorrhage may have ceased. Absence of the pulse does not necessarily indicate that the artery has been severed—it may be thrombosed or in spasm; nor does the development of a pulse exclude injury, as an arteriovenous fistula or false arterial aneurysm may later attest.

The proper management consists of formal exploration of the wound under a general anes-



FIG. 9. Traumatic (false) aneurysm of right subclavian artery after automobile accident. (case 4, see also fig. 10.)

thetic and primary suture of the arterial defect after debridement of its margins. Any associated nerve lesions must be sought for and managed according to the circumstances under which the patient is first seen and treated. If a wound is explored within the first several hours after injury, we not only perform primary repair of the artery, which is usually readily feasible, but we suture the nerves as well. If the nerves are not sutured at this time they are marked with either fine wire or black silk to facilitate the location of the ends when the patient is brought back to the hospital for anastomosis about 3 months later. In the instance of the 2-year-old child referred to previously, the ends of the completely severed brachial artery were found and anastomosed primarily with interrupted everting mattress sutures of 5-0 arterial silk. Pulsations were restored in the artery distal to the point of suture.

#### False Aneurysm (Pulsating Hematoma)

The false aneurysm is perhaps one of the most commonly mistreated traumatic arterial lesions. The physician frequently is aware that repeated hemorrhage has occurred, but at each investigation the artery is never formally exposed; thus

## ANEURYSM OF SUBCLAVIAN ARTERY

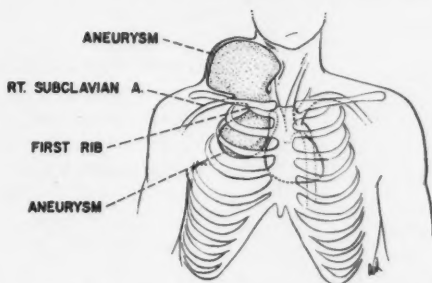


FIG. 10. This aneurysm, which followed blunt closed trauma, was approached by first gaining control of the innominate artery through a sternal splitting incision which was extended into the 3rd right interspace (case 4).

the old clot is considered to have resulted from the primary injury. Several weeks may pass before a definite pulsating hematoma discloses the presence of the false aneurysm (figs. 9 and 10).

#### Case 4. False Aneurysm (Pulsating Hematoma) of Right Subclavian Artery

A. P., a 58-year-old Negro man, was well until 1956 when he sustained fractures of the right clavicle and upper 6 ribs in an automobile accident. A few months after the accident he noticed a pulsating lump in the right supraclavicular fossa (fig. 9). This mass gradually enlarged until he felt that he had become weaker and was unable to walk the usual distance. At the time of admission to the University Hospital on May 11, 1959, a roentgenogram revealed that the aneurysm extended into the upper portion of the right hemithorax. It was considered that this was most likely a traumatic aneurysm, although it was not known whether it arose from the innominate, right common carotid, or right subclavian artery.

At operation on May 18, the first maneuver was to gain control of the innominate artery by splitting the sternum down to the 3rd rib and then veering into the right 3rd interspace. This permitted the elevation and retraction of a flap of the thoracic wall on the right to expose the great vessels of the upper mediastinum. After control of the proximal arterial supply had been obtained, the dissection was carried far up into the neck and a tape passed around the right common carotid artery distal to the aneurysm. Once this had been done it was felt that any back-bleeding from the subclavian artery could be controlled by compression, since it was not possible to expose the left subclavian artery until the aneurysm had been further mobilized. The dumbbell shape of

the aneurysm was due to the fact that it was compressed in its center by the unyielding bony structure of the thoracic inlet; little difficulty was experienced in freeing the lower portion from the right upper pulmonary lobe. The dissection was continued until finally it was possible to get around the aneurysm on all sides except posteriorly. Gradually it developed that the aneurysm arose entirely from the right subclavian artery just distal to its origin from the innominate artery. The major portion of the false aneurysm was removed, but it was not possible fully to excise the false sac posteriorly without running considerable hazard of injuring the brachial plexus, the phrenic nerve and other structures which could not be readily identified because of the densely adherent portions of the aneurysmal false sac. At this point a decision had to be made regarding the placing of an arterial prosthesis from the right common carotid artery or the innominate artery to the subclavian artery distally. In view of the friability of the right common carotid artery and the large amount of old sac remaining, it was decided to omit the placing of a prosthesis at that time, but to plan to return later for this simple procedure if the patient developed symptoms due to relative ischemia of the right arm.

The sternum was closed with several wire sutures and with interrupted silk.

Postoperatively he had several episodes of mild hypotension, but no definite etiologic factor was disclosed. Hypovolemia was not considered likely. The right radial pulse was weakly palpable the night of the operation and by the time of discharge on May 26, 1959, the right radial pulse was fair. When he was seen subsequently in the follow-up clinic, his wound was well healed, he had a right radial pulse, and he had had no symptoms referable to relative ischemia of the right arm.

### *Comment on False Aneurysms*

We have had several patients with false aneurysms of the arteries of the upper extremity. One was of the brachial artery; the short defect in the artery was excised, along with the false aneurysm, and ends of the artery were anastomosed. Since the portion of the arterial wall involved in a false aneurysm is usually small, often less than 1 cm. in length, this segment of the artery can often be excised, and the artery then repaired with continuous or interrupted sutures of arterial silk. At other times only one side of the artery is involved and this portion of the wall can be excised, leaving the normal side of the artery intact; the defect is closed transversely to avoid constriction of the lumen. False aneurysms may involve any artery of the arm, and we have excised such lesions of the radial and of the ulnar vessels. Shaw<sup>52</sup> has successfully anastomosed severed radial and ulnar arteries, but we have not yet had occasion to do so.

### *Compression and/or Spasm*

Arterial compression, with or without associated spasm,<sup>38</sup> may occur in any extremity. The best known example in the arm is the so-called Volkmann's ischemic compression with late contracture. The causes of arterial insufficiency include a variety of different pathologic circumstances, any of which may contribute to a reduction in blood flow through the brachial artery and ischemia of the distal extremity. First, severe hemorrhage in and about the elbow may result in increased pressure in a closed fascial space with resulting compression of the walls of the artery. Second, a cast or traction used to manage a fracture near the elbow may result in arterial compression, spasm, and/or thrombosis. Furthermore, the fracture fragments themselves may impinge against the artery. We were recently impressed with such an instance in the lower extremity, when the addition of a few pounds of weight to the balanced traction resulted in the reappearance of foot pulses and explained the cold and numb foot which had developed. It was apparent that the fragments of the tibial fracture were overriding and producing occlusion of the arteries supplying the foot. Third, there may be other direct injury to the artery itself, which is unrecognized because of the considerable swelling in the region of the elbow. Edwards and Lyons<sup>16</sup> have emphasized the importance of hemorrhage

beneath the arterial sheath, with displacement of the intima and media into the lumen to reduce blood flow. They recommend prompt surgical exploration in patients in whom arterial injury has resulted in absent pulses that are not restored by sympathetic nerve block. Fourth, severe arterial spasm<sup>10</sup> may develop. Regardless of the manner in which the normal volume of blood flow through the artery is reduced, however, the end result of ischemia is the same, namely a necrosis in the muscles of the forearm and hand. Actual gangrene of the skin of the hand may not occur (though it may), but there may be extensive muscle ischemia with fibrosis. Contracture may render the forearm and hand almost useless.

Volkman's ischemic contracture is usually avoidable by careful fracture reduction, and, thereafter, by frequent periodic examinations of the hand and radial pulse after the fracture has been reduced and the arm placed in a cast. If the patient complains of undue pain or the hand is cold and the radial pulse absent, the cast must be removed and efforts made to restore adequate blood flow to the distal extremity. This may require fasciotomy in the region of the elbow.

### *Thrombosis (Acute)*

Acute traumatic thrombosis of an artery may be produced in a variety of ways. Examples of closed trauma which may do this are the use of crutches, blunt force, and arterial injury associated with fractures and/or dislocations.<sup>30, 36, 45, 48</sup> In patients who have been injured by gunshot, particularly those who have had multiple lead pellets enter an extremity, it is not uncommon to find injury to the wall of a vessel with intramural hemorrhage. A bulging of intima into the lumen to occlude the vessel may occur. An arteriogram is frequently helpful but can be misleading, since stagnant blood may be present for some distance above the actual level of occlusion.<sup>35</sup> Nevertheless, the absence of a pulse distally should prompt exploration of the artery, particularly if paravertebral sympathetic blocks to relax spasm do not restore blood flow.

### *Arteriovenous Fistula*

Arteriovenous fistulas between the subclavian artery and the adjacent subclavian vein or the innominate vein are not rare. However, we recently had an opportunity to treat a patient who had an arteriovenous fistula between the



FIG. 11. Multiple arteriovenous fistulas due to shotgun injury (case 5). The use of a sterile stethoscope as a guide to small arteriovenous fistulas during operation, and to avoid leaving one untreated, is recommended.

right brachial artery and an adjacent antecubital vein.

*Case 5. Traumatic Arteriovenous Fistula between the Right Brachial Artery and an Adjacent Vein, in Association with a Second Arteriovenous Fistula in the Left Popliteal Space*

W. C., a 19-year-old white youth, was well until January 1959, when he was accidentally wounded with a shotgun blast which struck his right arm and both legs at close range. These wounds were satisfactorily managed in a hospital near his home, but in February he noticed a "vibration" and pain in the right antecubital fossa, and his family physician diagnosed an arteriovenous fistula.

On admission to the University Hospital on February 20, 1959, there was a definite thrill and a continuous bruit over the right antecubital fossa. An unexpected additional finding, however, was that of a bruit and thrill in the left popliteal fossa, representing a second arteriovenous fistula (fig. 11).

At operation on February 21, under general anesthesia, a transverse incision was made across the right antecubital fossa, and the brachial artery and adjacent structures were exposed. The palpable thrill was a ready guide to the site of the fistula between the distal extension of the brachial artery, just above its bifurcation into the radial and ulnar arteries, and a parallel vein. Tapes were passed around the proximal artery and around both of its branches just distal to the bifurcation, and similar tapes were passed around the vein above and below the site of the fistula (fig. 12). When this had been done all vessels were

temporarily occluded and the fistula was taken down by cutting squarely across the fistulous opening between the two vessels. This allowed one to visualize the small defect in each vessel and in each instance this defect was readily closed with a continuous suture of 5-0 arterial silk without occluding or encroaching upon the lumen of either vessel. This resulted in immediate restoration of a good pulse in the radial artery after release of the hernia tape, and blood was observed flowing readily through the vein and continuing proximally. A sterile stethoscope was used to make certain that no additional fistula remained. (Having had the disconcerting experience of missing a second arteriovenous fistula in a medical student who had been injured with birdshot in the right thigh while duck hunting, we have since employed a sterile stethoscope in all cases of arteriovenous fistula to make certain that a second fistula is not present.)

The wound in the right antecubital fossa was then closed, and the patient was turned on his face to afford an approach to the left popliteal fossa. This arteriovenous fistula was found to involve a small collateral arterial branch and vein, rather than being between the popliteal artery and the popliteal vein. It was a simple matter to excise the fistula. It appeared likely that the arteriovenous fistula in the right antecubital fossa and the one in the left popliteal fossa had each been caused by a single lead pellet.

Postoperatively the patient had a completely benign course and was discharged at the end of 5 days. All thrills and bruits were absent.

*Comment.* It has been possible to take down the

## REPAIR OF A-V FISTULA

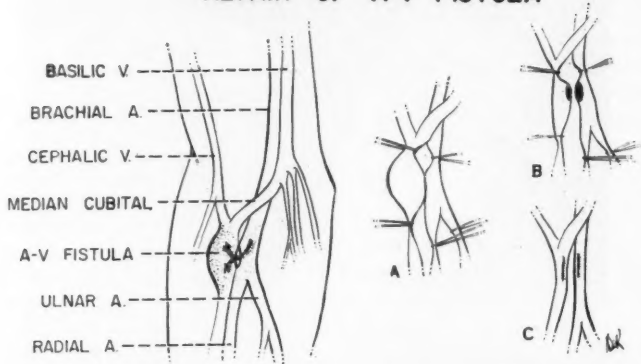


FIG. 12. Most arteriovenous fistulas can be taken down and the defect in both the artery and vein repaired after debridement of the margins. As a rule the vessels are best repaired transversely but often longitudinal closure is quite satisfactory (case 5).

fistula and close the defect in the artery in most cases of arteriovenous fistula treated in recent years; often the vein can be similarly repaired. The defect between the artery and the vein is usually small, and it can be managed either by excising the involved portion of the wall of the artery, with (usually) transverse closure, or with complete excision of the involved segment of the artery and then anastomosis of the ends of the divided artery. The same type of management can often be applied to the vein if the size of the vessel warrants repair.

#### *Concluding Remarks on Management of Arterial Trauma*

Despite the excellent collateral blood supply of the upper extremity, one should never be satisfied without considering the feasibility of an attempt to restore arterial continuity following an injury to the larger arteries of the arm. In analyzing 2471 cases of arterial injury in World War II, DeBakey and Simeone<sup>11</sup> found that interruption of the brachial artery above its bifurcation was followed by loss of a part of the extremity in one-fourth of the cases; if the artery was divided above the origin of the deep brachial artery, loss of some portion of the extremity occurred in almost 55 per cent of patients. The amputation rate was 39 per cent when both radial and ulnar arteries were divided, whereas ligation of the radial artery alone produced gangrene in 5 per cent of cases. It is well known that either the ulnar or the radial arterial arch in the hand may be reduced in importance or

virtually absent in some subjects. In a study of 408 upper extremities, Weathersby<sup>60</sup> found major arterial variants in 27.8 per cent.

The experience in the Korean War proved beyond all question the feasibility and value of immediate primary repair of injured arteries. Of 28 major arterial injuries above the brachial bifurcation, 25 were treated by immediate repair, and no amputations were required in this group.<sup>24</sup>

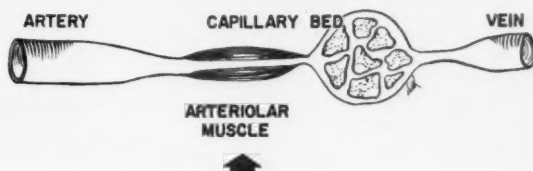
#### ANEURYSMS

Aneurysms due to syphilis or atherosclerosis may affect virtually all major arteries and those to the arm are not spared. Nevertheless, true aneurysms involving the subclavian, axillary or brachial artery are decidedly uncommon, in our experience. The general principles of management of a true aneurysm involving an artery of the upper extremity are the same as those presented in the discussion of false aneurysms. Although in many instances it would be feasible to resect the aneurysms with simple ligation of the vessels, the insertion of a graft should be considered in every case.

#### CONDITIONS INVOLVING THE SMALLER ARTERIES AND ARTERIOLES

The smaller arteries of the hands and fingers are often involved in one or another of a particularly intriguing group of diseases many of which are as yet poorly understood. In addition to certain dimly perceived lesions which have been omitted, the following will be discussed: Raynaud's disease, thromboangiitis obliterans, the vibrating

### THE ARTERIOLE



IMPORTANT  
IN RAYNAUD'S DISEASE &  
IN ARTERIOLAR SPASM  
ASSOCIATED WITH  
OTHER CONDITIONS

FIG. 13. The terminal arteries and the arterioles play important roles in the production of certain symptoms and physical findings found in various peripheral arterial diseases. Raynaud's phenomenon, which is observed not only in Raynaud's disease but in many other conditions as well, is caused by vasospasm. It may be observed in association with the scalenus anticus and cervical rib syndromes, thromboangiitis obliterans, atherosclerosis obliterans, brachial plexus injury and scleroderma, among others.

tool (percussion) syndrome, reflex neurovascular dystrophy, obliterative atherosclerotic disease, and other less well defined pathologic entities such as the syndrome of the middle-aged female and scleroderma. To these conditions will doubtless be added others as this particular group of diseases is further unraveled. For example, there can be no question but that many patients who were previously diagnosed as having thromboangiitis obliterans actually had occlusive disease of major arteries; many patients diagnosed as having Buerger's disease had had a sympathectomy for treatment of the presumed condition. Actually, atherosclerosis has been found to be the underlying pathology in many or most cases of Buerger's disease.

It has been particularly helpful, we think, to distinguish between true *Raynaud's disease* with pure spasm, at least in the earlier phases of the disease, and *Raynaud's phenomenon* which represents the ischemic changes due to spasm, whether the vasospasm occurs in true Raynaud's disease or as spasm superimposed upon a partial organic occlusion of the arteries to the digits. Thus Raynaud's phenomenon may be encountered in such separate entities as occlusion of the brachial artery with undue sensitivity of the fingers to cold because of the reduced blood flow, thromboangiitis obliterans, vibrating tool syndrome, and other conditions. Above all, when the patient exhibits evidence of ischemia of a finger one should no longer consider only Raynaud's disease or thromboangiitis obliterans, for there

are many circumstances, as we have seen, which can produce ischemic necrosis of the fingertip.

#### *Raynaud's Disease*

The underlying pathophysiology of Raynaud's disease<sup>46</sup> consists of an increased sensitivity of the smaller arteries and arterioles (fig. 13) of the hand to cold or to the sympathetic response to emotional crises.<sup>31-33</sup> The condition rarely occurs in males.<sup>21</sup> What is perhaps new in this field is that many patients formerly diagnosed rather loosely as having "Raynaud's disease" have since been found to have other more specific diseases such as early scleroderma, obliterative atherosclerotic disease, or a percussion trauma of the type found in pianists and typists.

**Treatment.** The treatment of Raynaud's disease has not varied particularly over the past few years. Conservative management often suffices. The patient is advised to avoid exposing the hands to cold or other trauma. Since chilling of the body will also result in vasospasm in the extremity, warm clothing must be worn. The use of Priscoline has been helpful upon occasion, given orally in doses of from 25 to 50 mg. 3 or 4 times daily. A woman of average size cannot usually take much more than 50 mg. of the drug every 3 hours without experiencing the chilly sensations which many patients find particularly annoying. When cervical sympathectomy<sup>2</sup> is required, most surgeons believe that the best and most lasting results are achieved by an

extensive procedure which removes the stellate ganglion and the first two ganglia below this level. This usually results in a Horner's syndrome. This is less objectionable if the sympathectomy is performed bilaterally with the production of symmetry even though bilateral Horner's syndrome is present. In contrast to the resection of the middle cervical (stellate) ganglion, plus the 1st, 2nd, and 3rd thoracic ganglia and their connecting sympathetic trunk, some surgeons still prefer to avoid the Horner's syndrome by performing a more limited operation. As for ourselves, we have tried to avoid sympathectomy as long as possible in patients with Raynaud's disease, but when the procedure has been resorted to, a Horner's syndrome has been accepted.

In some patients, unfortunately, the relief provided by sympathectomy is short-lived. Simeone<sup>55</sup> has examined this problem, including the nature of the supersensitivity which may develop in denervated digital blood vessels in man.

#### *Thromboangiitis Obliterans*

Thromboangiitis obliterans<sup>61</sup> or Buerger's disease<sup>9</sup> may prove to represent a manifestation of atherosclerosis. It usually involves the extremities only but may involve the viscera. The principal pathophysiology results from the ischemia of the tissues produced by occlusion of small and at times fairly large arteries such as the radial, ulnar, dorsalis pedis and posterior tibial. In contrast to Raynaud's disease, which occurs largely in the upper extremities and almost entirely in women, Buerger's disease is more commonly found in the lower extremities and usually, but not always,<sup>22</sup> in men. The segmental nature of the occlusion may permit considerable collateral circulation to develop. Superimposed vasospasm<sup>40</sup> (Raynaud's phenomenon) is often a prominent feature of the malady.

Although the etiology of Buerger's disease is disputed, it has been found to bear a relationship to smoking in many patients.<sup>37</sup> The vascular effects of smoking are widespread.<sup>56</sup>

A patient with thromboangiitis obliterans may show various degrees of necrosis of the fingers when the hands are involved. The following case study emphasizes many of the features of Buerger's disease and its management.

#### *Case 6. Thromboangiitis Obliterans (Possible) Involving the Hands*

S. M., a 45-year-old white man, was admitted to the University Hospital on June 1, 1956, with the history of having had a coronary occlusion in 1951 and an episode of cyanosis involving the left hand in 1953 which had cleared up spontaneously. The present urgent symptoms had begun about 6 months before admission. At that time he had begun to notice marked tingling, numbness, and cyanosis of all the fingers, but the symptoms of ischemic sensitivity to cold were most marked in the index finger of the right hand which blanched and became numb. This finger had continued to be the one most severely involved, but it had been found that all fingers blanched when the hands were washed in cool water. Moreover, numbness and tingling in various fingers often developed as he gripped the steering wheel while driving his car. Although once an inveterate smoker, he had stopped for several years after his coronary attack, but had begun to smoke rather excessively approximately 6 to 8 months previously, the approximate date of onset of the present symptoms. He had had no symptoms referable to the legs and feet.

On physical examination the various fingers of both hands gave evidence of mild trophic changes suggestive of relative ischemia, but only one finger, the index finger of the right hand, exhibited actual necrosis. It may be seen in figure 14 that other fingers, particularly the 5th finger of the right hand, were cyanotic at the time of examination. The radial pulses were present and of good quality. The blood pressure in both arms was equal and various changes in position of the right arm had no effect on the radial pulse, tending to exclude a scalenus anticus syndrome. Roentgenograms disclosed no cervical rib.

The diagnostic possibilities seemed to include mainly thromboangiitis obliterans and arteriosclerosis obliterans. The lack of symmetry, the presence of pain and evidence of ischemia even when the hands were warm, the apparent absence of emotional factors, the sex of the patient—all of these tended to exclude Raynaud's disease. Scleroderma was considered but was thought to be unlikely, particularly in view of the fact that the patient was a man. All things considered, the diagnosis of Buerger's disease seemed to be a tenable one.

An arteriogram was performed through the right radial artery (fig. 15). Both the ulnar and radial arteries were outlined, but occlusion of several of the digital arteries was demonstrated. Thus he did have occlusive disease of the small

Fig. 1.  
cyanosis  
instance

Fig.  
litanar  
arteries

vessels  
disease  
naud's  
superior  
Cour  
pain, p  
was be  
require  
was al

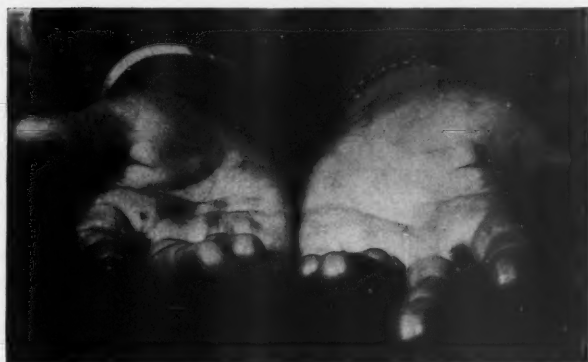


FIG. 14. Thromboangiitis obliterans (case 6). Note gangrene of tip of right index finger and the deep cyanosis of the right 5th finger. Thromboangiitis obliterans may represent atherosclerosis in most instances.



FIG. 15. Arteriogram in thromboangiitis obliterans (case 6). Note obliteration of most digital arteries.

vessels of the fingers, whether due to Buerger's disease or to arteriosclerosis obliterans. Raynaud's phenomenon,<sup>25</sup> due to vasospasm, was superimposed upon the organic occlusion.

*Course in hospital.* He was in almost continuous pain, particularly from his right index finger which was becoming necrotic at the tip, and opiates were required. A stellate ganglion block with procaine was also found to provide relief for the duration

of the effects of the medication, and thus a *right cervical sympathectomy* was performed on June 4, 1956. The middle cervical ganglion (stellate) and the 2nd and 3rd thoracic sympathetic ganglia were removed, the sympathetic chain being transected distal to the 3rd thoracic ganglion.

*Postoperatively* the pain in the right hand was much improved, and there was a progressive healing of the necrotic tip of the right index finger. He was discharged and was seen again on July 27, 1956, at which time the index finger had healed completely. During this time he had cut down on his smoking but had not stopped. He was urged to abandon smoking altogether because of both the digital and the coronary artery disease.

*Comment.* In the absence of a biopsy revealing the typical angiitis, this patient's disease was diagnosed as representing Buerger's disease largely by exclusion; atherosclerosis may exist. The relief of his pain by a stellate ganglion block was conspicuously effective, and thus sympathectomy was performed. The use of sympathectomy<sup>3</sup> and abstinence from smoking very commonly result in considerable benefit. We have not been particularly impressed with the value of vasodilator agents in improving blood flow through the fingers or toes and in reducing the pain which these patients have. The present patient was well on his way to opiate addiction at the time his sympathectomy was performed to give relief from the throbbing pain caused by ischemia.

#### *The Vibrating Tool (Percussion) Syndromes*

In 1918 Hamilton<sup>20</sup> pointed out the effects which the use of an air hammer had produced in

the hands of stone cutters. Since then numerous reports have emphasized the fact that repeated percussion trauma may result in an increased sensitivity of the hands to cold resulting in Raynaud's phenomenon. This "local fault" is the result of long repeated trauma which causes changes in the intima and the walls of the arteries, with reduction in the size of the lumen in some instances. This so-called "vibrating tool syndrome" is often relatively mild and some patients may be able to continue their usual work without particular therapy. Similar changes have been described in pianists and in typists.<sup>6</sup> Thus the term "vibrating tool syndrome" is a misnomer, for the injury is actually due to repeated percussion rather than to vibration *per se*.<sup>5</sup> It is the fingers primarily involved with holding the pneumatic drill, or those involved with typing and piano playing, that are altered. Frequently a single finger may be affected and the others spared. However, in the case of heavy instruments such as the pneumatic drill the process gradually spreads to fingers more distant from those which are primarily used to grip the instrument.

**Management.** By and large, these syndromes respond to rest or to a change of jobs, and they may be tolerated indefinitely by some individuals who are able to continue their usual work. The following case probably represents the occurrence of such a condition.

#### *Case 7. Percussion Syndrome in a Typist*

M. P., a 33-year-old white lady, had long served as the typist in a team with her husband in the operation of a printing shop. Three years before her first examination by the author in October 1959, she had noticed numbness and tingling in the right middle finger but occasionally in other fingers as well. This finger gradually became cooler than the other fingers and often was numb and blue, being quite sensitive to cold. It produced a throbbing pain from time to time, and some nights she was unable to sleep because of this. There slowly developed an indolent point of brownish discoloration of the tissue adjacent to the radial surface of the tip of the fingernail. Subsequently this area became infected.

On physical examination it was found that all fingers seemed to be within normal limits except for the right middle finger. This digit was definitely cool, slightly swollen, and moderately cyanotic, and there was excessive growth of tissue beneath the distal portion of the fingernail, with a site of impending necrosis as described above. The radial pulse was excellent on both sides and

was not altered by changes in posture. The blood pressures were equal in the two arms, and it appeared unlikely that cervical rib compression or occlusive disease existed.

**Subsequent course.** The most tenable diagnosis appeared to be a percussion syndrome caused by typing. Her age of 33 weighed against atherosclerotic occlusive disease, the lack of symmetry appeared to rule out Raynaud's disease, and Buerger's disease is decidedly uncommon in women and above all in women who have never smoked.

A plan of conservative management was instituted. The patient was instructed carefully in the avoidance of exposure of her hands to heat, cold or trauma. Further use of the right middle finger in typing was to be avoided, although she was permitted to use the apparently normal fingers to operate an electrical typewriter. Priscoline hydrochloride was prescribed in a dosage of 50 mg. orally every 3 hours when awake. Initially this dosage produced some gastrointestinal disturbances as well as sensations of marked chilliness, and the amount was temporarily reduced to 25 mg. every 3 hours. However, presently she was able to increase the dosage again to 50 mg. every 3 hours and this was then maintained. This regime produced definite improvement in the involved finger. The matter of right cervical sympathectomy was considered and was discussed with the patient. Yet as of this writing it appears that this will not be necessary. She can now sleep through the night without being awakened by ischemic throbbing pain.

Actually, sympathectomy has not often been required in patients with the percussion syndrome. Inasmuch as this patient's disease thus far is unilateral, should a sympathectomy be required an effort will be made to avoid a Horner's syndrome.

#### *Reflex Neurovascular Dystrophy*

Brief mention should be made of the group of obscure processes which are referred to collectively as instances of reflex neurovascular dystrophy.<sup>13, 17</sup> Such varied conditions as myocardial infarction, cold injury to the extremity,<sup>8, 20</sup> traumatic injury to the region of the brachial plexus, and gunshot wounds to the arteries of the arm with associated *causalgia*<sup>12, 24</sup> may all produce this reflex sympathetic phenomenon. The syndrome may include numerous symptoms, but the more prominent among them are vasomotor disturbances with persistent coldness, excessive sweating, cyanosis, and occasionally Raynaud's phenomenon; there may be edema, atrophy, hyperesthesia and paresthesia, and often severe burning pain with "trigger points" the stimulation of which may exacerbate markedly the

distress. Neurovascular dystrophy may be associated with an extensive demineralization of the associated bones, over and above what might be caused by disuse (Sudeck's osteoporosis).<sup>57</sup>

The many different conditions which may result in neurovascular dystrophy attest its complexity. Nevertheless, perhaps the single most common feature of the various clinical pictures is the evidence of sympathetic nervous system dysfunction. This "shoulder-hand" syndrome is very similar in many respects to the typical causalgia-type pain affecting the lower extremity secondary to a gunshot wound which need not have severed a major artery or nerve. The pain is often relieved temporarily by cervical sympathetic block, and in many cases cervical sympathectomy has been employed. In fact, as regards causalgia in the lower extremity after trauma, almost by definition this pain must be relieved by interruption of the sympathetic nerve supply to the leg if the diagnosis of causalgia is to be considered established. The fact that a more extensive experience with sympathectomy for painful conditions of the upper extremity does not exist is the fact that most surgeons wish to avoid the Horner's syndrome where possible, in addition to the slightly more difficult operative approach required for cervical sympathectomy.

We have seen several instances of severe shoulder-hand syndrome, the most conspicuous one having followed traumatic injury to the right shoulder in a young man. As a rule conservative measures, consisting of stellate ganglion blocks, reassurance, and physical therapy, have allowed us to avoid cervical sympathectomy.

#### *Obliterative Atherosclerotic Disease*

Little need be said regarding the arteriosclerosis obliterans that may involve the finer vessels of the hands and fingers. Atherosclerosis may affect any artery of the body, and fingers are no exception. When the condition exists, all the usual conservative measures—including vasodilator drugs, avoidance of trauma to the digits, and avoidance of extremes of heat or cold—are to be used. If these measures prove insufficient to control the ischemic pain, cervical sympathetic block may be employed and, if temporary relief is provided, cervical sympathectomy may be employed in the selected case. It is only occasionally that one will be forced to amputate a digit because of this condition, since



FIG. 16. Extensive gangrene of fingers due to scleroderma. There is considerable evidence that scleroderma may be the result of vascular disease. The development of scleroderma after "Raynaud's disease" of years' standing was remarked by Jonathan Hutchinson.<sup>23</sup>

collateral blood supply usually suffices to maintain viability even if not full use of the finger.

#### *The Syndrome of the Middle-Aged Woman*

Jepson<sup>25</sup> has called attention to a group of female patients who have evidence of organic disease of the digital arteries with superimposed Raynaud's phenomenon. The precise etiology is not clear, since it occurs too late in life to be primarily inborn and there is no evidence of a diffuse atherosclerosis. The one feature which is relatively common is that it occurs near the menopause and that reestablishment of the menses, either with estrogen therapy or spontaneously, has often been associated with improvement. This particular entity is mentioned here in order to take note of a rather nebulous group of peripheral arterial conditions which are as yet poorly understood.

#### *Scleroderma*

Scleroderma may progress to a point in which it produces gangrene of the tips of most of the digits<sup>51</sup> (fig. 16), but its etiology is obscure. It is felt by some that it may represent the end result of disease of the smaller arteries; that eventually scleroderma may be demonstrated to represent primarily a vascular disease. For example, patients with Raynaud's disease for many years may eventually develop scleroderma.<sup>5, 23</sup> There is always the hazard, moreover, that the patient, particularly a woman, who is diagnosed as having Raynaud's disease or obliterative atherosclerotic disease may in fact ultimately prove to have or develop scleroderma. In biopsies taken from

patients with scleroderma it has been found that there is an increase in fibrous tissue and a loss of elastic tissue in the vessel walls, with gradual encroachment upon the lumen. No truly effective treatment is known.

*Concluding Comment on Conditions  
Involving the Terminal Arteries of the  
Hands and Fingers*

The primary objective in discussing here this group of conditions has been to emphasize the importance of continued careful observation by the physician as he sees cases which may fall into various of these groups. During recent years there has been a continuous unfolding and better differentiation of these diseases that result in ischemia of the tips of the fingers. Undoubtedly many additional etiologic circumstances will be delineated in the future.

SUMMARY

1. Arterial lesions of the upper extremity are in general representative of arterial lesions elsewhere, but certain of these are peculiar to the arm.

2. Major gangrene is not often associated with arterial diseases of the upper extremity, since the collateral circulation is extensive. Nevertheless, such diseases can produce far more physical disability than is commonly appreciated.

3. The purpose has been to emphasize the range and in some circumstances the complexity of subclavian, axillary, brachial and more distal arterial pathology.

4. The following conditions have been reviewed: Costocervical outlet syndromes with subclavian artery compression, occlusive disease of major arterial divisions, embolism, traumatic lesions of the larger arteries, aneurysms, and conditions such as Raynaud's disease, thromboangiitis obliterans, vibrating tool percussion syndromes, reflex neurovascular dystrophy, obliterative arteriosclerotic disease of the digital arteries, and other less clearly defined entities.

*Department of Surgery  
University of Mississippi Medical Center  
Jackson, Mississippi*

REFERENCES

1. ADSON, A. W., AND ALLEN, E. V.: Vascular clinics. VI. Thrombosis of arteries of the right upper extremity resulting from anomalous first rib. Proc. Staff Meet. Mayo Clin., 13: 637, 1938.
2. ADSON, A. W., AND BROWN, G. E.: The treatment of Raynaud's disease by resection of the upper thoracic and lumbar sympathetic ganglia and trunks. Surg., Gynec. & Obst., 48: 577, 1929.
3. ADSON, A. W., AND BROWN, G. E.: Thromboangiitis obliterans; results of sympathectomy. J. A. M. A., 99: 529, 1932.
4. ADSON, A. W., AND COFFEY, J. R.: Cervical rib; a method of anterior approach for relief of symptoms by division of the scalenus anticus. Ann. Surg., 85: 839, 1927.
5. ALLEN, E. V., BARKER, N. W., AND HINES, E. A., JR.: *Peripheral Vascular Diseases*, Ed. 2. W. B. Saunders Company, Philadelphia, 1955.
6. BARKER, N. W., AND HINES, E. A., JR.: Arterial occlusion in the hands and fingers associated with repeated occupational trauma. Proc. Staff Meet. Mayo Clin., 19: 345, 1944.
7. BEYER, J. A., AND WRIGHT, I. S.: The hyperabduction syndrome, with special reference to its relationship to Raynaud's syndrome. Circulation, 4: 161, 1951.
8. BLACKWOOD, W.: Studies in pathology of human "immersion foot." Brit. J. Surg., 29: 329, 1944.
9. BUEGERER, L.: *The Circulatory Disturbances of the Extremities; Including Gangrene, Vasomotor and Trophic Disorders*. W. B. Saunders Company, Philadelphia, 1924.
10. COHEN, S. M.: Traumatic arterial spasm. Lancet 1: 1, 1944.
11. DEBAKEY, M. E., AND SIMEONE, F. A.: Battle injuries of the arteries in World War II. An analysis of 2471 cases. Ann. Surg., 123: 534, 1946.
12. DETAKÁTS, G.: Causalgic states in peace and war. J. A. M. A., 128: 699, 1945.
13. DETAKÁTS, G.: Reflex dystrophy of the extremities. Arch. Surg., 34: 939, 1937.
14. DONALD, J. M., AND MORTON, B. F.: The scalenus anticus syndrome with and without cervical rib. Ann. Surg., 111: 709, 1940.
15. EDEN, K. C.: The vascular complications of cervical ribs and first thoracic rib abnormalities. Brit. J. Surg., 27: 111, 1939.
16. EDWARDS, W. S., AND LYONS, C.: Traumatic arterial spasm and thrombosis. Ann. Surg., 140: 318, 1954.
17. EVANS, J. A.: Reflex sympathetic dystrophy; report on 57 cases. Ann. Int. Med., 26: 417, 1947.
18. FREEMAN, N. E.: Acute arterial injuries. J. A. M. A., 139: 1125, 1949.
19. HAIMOVICI, H.: Peripheral arterial embolism; A study of 330 unselected cases of embolism of the extremities. Angiology, 1: 20, 1950.
20. HAMILTON, A.: Effect of the air hammer on the hands of stonecutters. Pub. Health Rep., 33 (part 1): 488, 1918.
21. HINES, E. A., JR., AND CHRISTENSEN, N. A.: Raynaud's disease among men. J. A. M. A., 129: 1, 1945.
22. HORTON, B. T., AND BROWN, G. E.: Thromboangiitis obliterans among women. Arch. Int. Med., 50: 884, 1932.
23. HUTCHINSON, J.: Acro-scleroderma following Raynaud's phenomena. Clin. J., 7: 240, 1896.
24. JAHNKE, E. J., JR., AND SEELEY, S. F.: Acute

- vascular injuries in the Korean War. An analysis of 77 consecutive cases. *Ann. Surg.*, 138: 158, 1953.
25. JEPSON, R. P.: Raynaud's phenomenon—a review of the clinical problem; Hunterian lecture. *Ann. Roy. Coll. Surgeons, England*, 9: 35, 1951.
  26. JONES, F. W.: The anatomy of cervical ribs. *Proc. Roy. Soc. Med. (Clin. Sect.)*, 6: 95, 1912-1913.
  27. KLEINBERG, S., AND LEVINE, M. A.: Headache as a symptom of cervical rib. *Ann. Surg.*, 105: 299, 1937.
  28. KÜTTNER, H., AND BARUCH, M.: Der traumatische segmentäre Gefäßkrampf. *Beitr. klin. Chir.*, 120: 1, 1920.
  29. LEARMONTH, J. R.: Some sequels of abnormality at the thoracic outlet. *Thorax*, 2: 1, 1947.
  30. LEFEVRE, F. A.: Arterial thrombosis following simple contusion; report of a case. *Am. Heart J.*, 17: 111, 1939.
  31. LEWIS, T.: Raynaud's disease, with special reference to the nature of malady. *Brit. M. J.*, 2: 136, 1932.
  32. LEWIS, T., AND LANDIS, E. M.: Further observations upon a variety of Raynaud's disease with special reference to arteriolar defects and to scleroderma. *Heart*, 16: 329, 1951.
  33. LEWIS, T., AND PICKERING, G.: Observations upon maladies in which the blood supply to digits ceases intermittently or permanently and upon bilateral gangrene of digits; observations relevant to so-called "Raynaud's disease". *Clin. Sc.*, 1: 327, 1934.
  34. LINDSKOG, G. E., AND HOWES, E. L.: Cervical rib associated with aneurysm of the subclavian artery; report of a case and review of the recent literature. *Arch. Surg.*, 34: 310, 1937.
  35. LOGAN, W. D., AND GOUELOCK, W. J.: Arteriographic errors in localization of acute arterial obstruction; case report and experimental studies. *Surgery*, 43: 974, 1958.
  36. LOWENBERG, E. L.: Acute traumatic arterial thrombosis of the extremities. *Virginia M. Monthly*, 67: 630, 1940.
  37. MADDOCK, W. G., MALCOLM, R. L., AND COLLIER, F. A.: Thrombo-angiitis obliterans and tobacco; the influence of sex, race, and skin sensitivity to tobacco on cardiovascular responses to smoking. *Am. Heart J.*, 12: 46, 1936.
  38. MONTGOMERY, A. H., AND IRELAND, J.: Traumatic segmentary arterial spasm. *J. A. M. A.*, 105: 1741, 1935.
  39. MONTGOMERY, H.: Experimental immersion foot. Review of the physiopathology. *Physiol. Rev.*, 34: 127, 1954.
  40. MORTON, J. J., AND SCOTT, W. J. M.: Some angiospastic syndromes in the extremities. *Ann. Surg.*, 94: 839, 1931.
  41. MURPHY, T.: Brachial neuritis caused by pressure of first rib. *Australian M. J.*, 16: 582, 1910.
  42. NAFFZIGER, H. C.: Editorial. The scalene syndrome. *Surg. Gynec. & Obst.*, 64: 119, 1937.
  43. NEWELL, R. L.: Cervical rib with vascular complications. *Brit. M. J.*, 1: 782, 1933.
  44. OCHSNER, A., G. MAGE, M., AND DEBAKEY Scalenus anticus (Naffziger) syndrome. *m A. J. Surg.*, 28: 669, 1935.
  45. PLATT, H.: Occlusion of the axillary artery due to pressure by a crutch; report of two cases. *Arch. Surg.*, 20: 314, 1930.
  46. RAYNAUD, A. G. M.: *De l'Asphyxie Locale et de la Gangrène Symétrique des Extrémités*. Rignoux, Paris, 1862.
  47. ROB, C. G., AND STANDEVEN, A.: Arterial occlusion complicating thoracic outlet compression syndrome. *Brit. Med. J.*, 2: 709, 1958.
  48. ROB, C. G., AND STANDEVEN, A.: Closed traumatic lesions of the axillary and brachial arteries. *Lancet*, 7: 597, 1956.
  49. ROSS, J. P.: The vascular complications of cervical rib. *Ann. Surg.*, 150: 340, 1959.
  50. SCHEIN, C. J., HAIMOVICI, H., AND YOUNG, H.: Arterial thrombosis associated with cervical ribs: surgical considerations; report of a case and review of the literature. *Surgery*, 40: 428, 1956.
  51. SELLEI, J.: Die Akrosklerosis (Sklerodaktylie) und deren Symptomenkomplex nebst neuen Untersuchungen bei Sklerodermie. *Arch. Dermat. u. Syph.*, 163: 343, 1931.
  52. SHAW, R. S.: Reconstructive arterial surgery in upper-extremity injuries. *J. Bone & Joint Surg.*, 41-A: 665, 1959.
  53. SHENKIN, H. A.: Cervical rib and thrombosis of the subclavian artery. *J. A. M. A.*, 165: 335, 1957.
  54. SHUMACKER, H. B., JR.: Causalgia. III. A general discussion. *Surgery*, 24: 485, 1948.
  55. SIMEONE, F. A., AND FELDER, D. A.: The supersensitivity of denervated digital blood vessels in man. *Surgery*, 30: 218, 1951.
  56. Smoking and the cardiovascular system (editorial). *J. A. M. A.*, 150: 1016, 1952.
  57. SUDECK, P.: Über die akute (reflektorische) knochenatrophie nach Entzündungen und Verletzungen an den extremitäten und ihre klinischen Erscheinungen. *Fortschr. a.d. Geb. d. Röntgenstrahlen*, 5: 277, 1901-1902.
  58. TELFORD, E. D., AND MOTTERSHEAD, S.: Pressure at cervico-brachial junction; an operative and anatomical study. *J. Bone & Joint Surg.*, 30-B: 249, 1948.
  59. TELFORD, E. D., AND STOFFORD, J. S. B.: The vascular complications of cervical rib. *Brit. J. Surg.*, 18: 557, 1931.
  60. WEATHERSBY, H. T.: Anomalies of brachial and antebrachial arteries of surgical significance. *South. M. J.*, 49: 46, 1956.
  61. VON WINIWARTER, F.: Über eine eigentümliche Form von Endarteritis und Endoplebitis mit Gangran des Fusses. *Arch. Klin. Chir.*, 23: 202, 1879.
  62. WISON, S. A. K.: Some points in the symptomatology of cervical rib, with especial reference to muscular wasting. *Proc. Roy. Soc. Med.*, 6 (Clin. Sect.): 133, 1912-1913.
  63. WRIGHT, I. S.: The neurovascular syndrome produced by hyperabduction of the arms; the immediate changes produced in 150 normal controls, and the effects on some persons of prolonged hyperabduction of the arms, as in sleeping, and in certain occupations. *Am. Heart J.*, 29: 1, 1945.

## SURGICAL TREATMENT OF HYPERPARATHYROIDISM\*

FRANK GLENN, M.D.

New York, New York

In 1930 Eugene DuBois<sup>10</sup> established the diagnosis of hyperparathyroidism for the first time in the United States. The patient, Charles Martel, was subjected to repeated surgical explorations at the Massachusetts General Hospital which led to the demonstration and removal of a parathyroid adenoma from the mediastinum. The experience of Churchill and Cope<sup>2</sup> with 11 patients from whom parathyroid adenomas had been removed was published in 1934 and stimulated wide interest in the natural history of the condition as well as in the methods of diagnosis and the problems of surgical treatment. Since then several hundred patients have been successfully treated surgically for hyperparathyroidism. Although the largest series reported has been from the Massachusetts General Hospital and the Mayo Clinic, numerous sizable groups have been recorded from many other medical centers.

Hyperparathyroidism is due to an excess of Parathormone. This hormone produced by the parathyroid tissue is specifically concerned with the metabolism of calcium and phosphorus. Its exact mode of action is open to question. One theory holds that when present in excess of the normal it decreases the reabsorption of phosphorus in the renal tubule causing it to be lost in the urine and lowers its concentration in the blood. As the phosphorus content of the blood is reduced the calcium is increased. In their ionized forms calcium and phosphorus are maintained in the blood serum at a constant total value. Thus as phosphorus is decreased in the blood serum the calcium requirement mounts and is derived chiefly from bone. The increased calcium in the blood serum tends to be lost through the kidney into the urine. Thus excess Parathormone causes hypophosphatemia, phosphaturia, hypercalcemia and hypercalciuria and gradually depletes the calcium reserve of the body. Another concept supported by experimental work in animals whose kidneys have been removed maintains that Parathormone acts directly upon the bone liberating calcium into the circulation.

\* From the Department of Surgery, The New York Hospital-Cornell Medical Center New York 21, New York.

*Clinical symptoms.* In our experience calculi in the urinary tract are an early manifestation of hyperparathyroidism. The hyperactivity of the parathyroid tissue liberates an excess of Parathormone into the circulation. This acts upon the fixed calcium of the body resulting mainly in a decalcification of the bones. The calcium thus freed into the blood stream is excreted by the kidneys in the urine. As this occurs calcium may be deposited within the renal tissue or be precipitated from the urine to form calculi in the kidney-pelvis, the ureters or bladder. Skeletal changes appear much later as a rule and are a manifestation of prolonged liberation of calcium from the bones. The degree of hyperparathyroidism and the rapidity with which the freeing of calcium from the body stores occurs, determines the course of the disease. For example, only slight elevation of Parathormone might well be compensated for long periods whereas a sudden marked elevation might result in urinary calculus formation and nephrocalcinosis. If Parathormone is present in the blood stream in large amounts for prolonged periods as in carcinoma of the parathyroid with metastatic spread, then the calcium concentration may be sufficient to result in its being deposited throughout the entire vascular system, the pancreas, stomach and soft tissues. Under these circumstances osteoporosis is marked. Hypercalcemia and hypercalciuria are preceded by low serum phosphorus levels which persist as long as the hyperparathyroidism.

*Anatomy.* The normal parathyroid gland has the shape of a pea, is small, measuring about 4 by 3 by 1 mm and weighs less than half a gram. It varies greatly in color, from mahogany brown to gray yellow, and has an intermingling of fat. A thin but distinct capsule renders its dissection from adjacent tissue easy. In cross-section its texture is homogeneous and resembles thyroid tissue, but the color is often a distinctive pink.

Usually there are 4 parathyroid glands situated posterior to the thyroid lobes. The superior parathyroids are either near the middle and upper third, posterior to the thyroid lobe, or along the branches of the superior thyroid artery. However, their major blood supply is from the inferior thy-

roid artery. The inferior parathyroids rest near the inferior portion of the thyroid lobe posteriorly. Their blood supply is also from the inferior thyroid. The upper parathyroids are derived embryologically from the 4th branchial pouches. The lower two, together with the thymus, arise from the 3rd branchial pouches. Their origin helps to explain the aberrant position in which both normal and tumorous parathyroid glands are found. The upper parathyroids are generally more constant in their position, whereas the lower ones are not infrequently associated with the thymus in the anterior mediastinum.

Hyperparathyroidism may be the result of diffuse hyperplasia of all the parathyroid glands, an adenoma arising from one or more of them, or a carcinoma of parathyroid tissue. When there is hyperplasia of all the parathyroids, the normal anatomic relations usually exist. In about 20 per cent of adenomas, the parathyroid from which it arises is in an abnormal location. Perhaps the same is true for carcinomas of the parathyroid. In hyperplasia the glands may be enlarged to many times the normal and may vary somewhat one from another.

In contrast to the hyperplastic parathyroid gland is the benign adenoma which varies greatly in appearance from that of a slightly enlarged normal gland in its common position to an irregular nodular tumor weighing up to several grams. In shape they may be elongated and cylindrical or spherical. The majority of adenomas are found in the region of the normally placed parathyroid glands but they may be within the substance of the thyroid gland or the thymus, or they may be located at a level from just above the origin of the superior thyroid arteries to below the aortic arch. Most often they are brown, orange yellow, or pink in color. The larger tumors are frequently cystic.

Carcinoma<sup>5, 7</sup> of the parathyroid originates in one gland and tends to metastasize by direct extension or via the lymphatics and the blood stream. The degree of hyperparathyroidism produced by carcinoma is usually much greater than that resulting from benign adenomas, particularly those of relatively short duration. The intense hyperparathyroidism or carcinoma of one parathyroid almost without exception causes marked atrophy of the remaining glands.

Recently established severe hyperparathyroidism or moderate hyperparathyroidism of long standing is usually associated with osteoporosis

and bone cyst formation. These are readily demonstrated by x-ray. The partially decalcified bone is the site of changes other than the loss of calcium. There appear osteoblasts with hemorrhage followed by fibrosis. This degeneration of bone, if localized, leads to cyst formation. If these cysts are numerous or large, they destroy the normal bone architecture and so weaken its structure that spontaneous or pathologic fractures occur.

Hypercalcemia occurs in a number of conditions that require consideration in a differential diagnosis. These include renal rickets, bone atrophy due to disease or disuse, hypervitaminosis D, milk-alkali syndrome, osteolytic metastatic carcinoma, multiple myeloma, and sarcoidosis. It is to be borne in mind that hyperparathyroidism may be secondary to disease elsewhere. Renal rickets is the best example. The kidney excretes excessive calcium and the parathyroids are called upon to provide additional Parathormone to mobilize more calcium to meet the demand. However, since the diagnosis is based essentially on the presence of hypercalcemia other causes of hypercalcemia must be excluded. If hypercalcemia due to other etiologic factors is methodically excluded, the differential diagnosis should not be particularly difficult. Sarcoid was formerly particularly troublesome. It has been learned recently that if hypercalcemia is associated with sarcoid, the value for calcium reverts to normal promptly after cortisone or adrenocorticotrophic hormone (ACTH) is administered.<sup>1</sup> Hypercalciuria accompanies all hypercalcemic conditions except that due to excessive calcium ingestion. Hypercalcemia alone does not justify the diagnosis of hyperparathyroidism, but whenever demonstrated should be investigated. Its presence is of serious import, and the correct diagnosis must be established.

Clinical hyperparathyroidism may be due to adenoma, hyperplasia or carcinoma of parathyroid tissue. In a fair proportion of patients, perhaps 10 per cent, there are abnormalities in other endocrine glands. These include the thyroid, pancreas, pituitary and adrenal. Cope and his co-workers have pointed out that the incidence of associated endocrine abnormalities is greater among those with hyperplasia of parathyroid glands. However, associated endocrine abnormalities have also been reported with adenomas of one or more of the parathyroid glands. Islet cell tumors of both the insulin and noninsulin producing type have been reported.

Peptic ulcer occurs sufficiently frequently with hyperparathyroidism that the astute clinician looks for the one when confronted with the other. St. Goar<sup>15</sup> estimates that from 9 to 15 per cent of patients with hyperparathyroidism have an associated peptic ulcer. Over 60 per cent of these peptic ulcers are duodenal, perhaps less than 25 per cent are gastric, and in the remainder both duodenal and gastric ulceration is present. The reported incidence from the Massachusetts General Hospital<sup>4</sup> and the Johns Hopkins group<sup>11</sup> is in agreement with St. Goar.

The pathogenesis, says Kirsner<sup>13</sup> of Chicago, is uncharted. A genetic explanation has been suggested because of family groups with involvement of parathyroid and other endocrine glands with peptic ulcer. Underdahl and associates<sup>16</sup> reported 8 patients with multiple adenomas of the parathyroids, the pituitary, and pancreatic islets, 3 of whom had peptic ulcers. Wermer<sup>17</sup> reported a family, father and 4 siblings, with a similar combination of polyglandular involvement. Of these 5, 4 had peptic ulcer.

Another example is reported by Kirsner.<sup>13</sup> The patient, a 38-year-old Negro, had recurrent peptic ulceration despite medical management, vagotomy and gastroenterostomy and gastric irradiation, and died from hemorrhage due to peptic esophagitis; at postmortem 2 large parathyroid adenomas were found, a basophilic adenoma of the pituitary and a small alpha cell tumor of the pancreatic islets.

In 1953, Elkeles<sup>6</sup> reported in *Lancet* a 46-year-old patient with advanced hyperparathyroidism who had marked osteoporosis with pathologic fractures who was demonstrated to have both a duodenal and a gastric ulcer. She refused operation and died a few weeks later. No postmortem was done.

During the past decade several reports<sup>8, 14</sup> have been published describing pancreatitis of the acute and recurrent type associated with hyperparathyroidism. Coffey and associates<sup>8</sup> collected 10 patients with chronic pancreatitis with calcification. Hoar and Gorlin<sup>12</sup> in discussing hyperparathyroidism and acute pancreatitis suggest 2 possible mechanisms in the involvement of the pancreas. The first of these is attributed to focal pancreatic necrosis caused by excess Parathormone and the second to the thought that hypercalcemia produces increased calcium in the external excretion of the pancreas. This is precipi-

tated in the pancreatic juices. We have observed pancreatitis in only 2 patients in our series. It is probable that in the past we overlooked this complication or associated condition because we have been searching for it only in recent years.

**Operation.** Careful planning is essential to success in the surgical removal of lesions causing hyperparathyroidism. Provision for evaluating tissue by the surgical pathologist during the operation is important. Likewise the time requirement for a meticulous search for an elusive tumor is unpredictable and the procedure should be approached with this in mind.

Under endotracheal anesthesia the thyroid gland is exposed as completely as possible in order to facilitate inspection of the four poles simultaneously. Any difference in color, size, vascularity of any one area as compared with another area of the thyroid gland may be significant. The extremities of the thyroid gland, the upper and lower poles, are the common sites for adenomas, and slight distortion or variation in vascularity may indicate the location of a tumor. If a lead is thus gained as to the location of an adenoma, it is well to explore this area first. If there is no indication, then a systematic dissection is done, beginning with the isolation and then the division of the vessels supplying the upper pole on the right, being sure that any aberrant blood vessels are identified.

After division of the lateral thyroid vein, the lower pole is exposed, and the recurrent laryngeal nerve demonstrated along the trachea. With the right lobe reflected medially the posterior surface is inspected. The parathyroids are identified if present first at the upper pole and then the lower. Normally there is a parathyroid in the vicinity of each pole. If absent, then it is an indication that an abnormally placed parathyroid is likely and that it may be an adenoma. The left lobe is inspected next.

In all patients the carotid arteries are examined for aberrant branches. If any are present, they are traced to their terminations. Sometimes these lead one to a parathyroid adenoma.

Thymus tissue is frequently encountered projecting up from the anterior mediastinum in the region of the inferior thyroid artery. Because the thymus and the inferior parathyroids are derived from the 3rd branchial cleft, it is not unusual to find these tissues intermingled. The parathyroid adenomas we have found in thymus tissue have

had their blood supply from the inferior thyroid artery. The dissection of the lower pole of the thyroid should be carried out with its numerous possible anatomic variations in mind. No structure should be divided until its origin and termination have been established. When both upper and lower poles have been well mobilized or divided, the thyroid lobe is readily reflected away from the trachea up to the isthmus. This permits one finally to re-evaluate it by palpation for additional parathyroid tissue on its posterior surface or within its substance. We have not observed parathyroid tissue in or beneath the thyroid isthmus.

When an adenoma is found there is a tendency to abandon further dissection. The surgeon should discipline himself to be complete and thorough in his examination at operation of all areas where a tumor may be found. Hence both lobes are meticulously dissected and evaluated. About 10 per cent of patients with hyperparathyroidism have more than one tumor; therefore, the exploration should be complete.

*Clinical material.* Over a period of 23 years (1937 to 1960) among a group of 46 patients with primary hyperparathyroidism at The New York Hospital we have demonstrated by operation the causes to be as follows:

Benign adenoma.....	38
Hyperplasia.....	5
Carcinoma.....	2
? Adenoma ? Carcinoma.....	1
	<hr/> 46

In the group there were 25 female patients and 21 male patients. A girl, 15 years of age who presented with ureteral colic as her initial complaint, was the youngest patient whereas a man, 66 years old, was the oldest. Two or more operations were performed in 5 patients before a tumor was found. It was situated in the normal position of the parathyroid in 37 instances, within the thyroid substances in 3 and below the level of the clavicles in the mediastinum in 4.

During this period we operated upon 2 other patients in whom we could not locate the adenoma. Subsequently both were operated upon by Dr. Oliver Cope of Boston, who removed an adenoma. In one of the 46 patients being reported, he demonstrated an adenoma that I had

not been able to find in a patient at The New York Hospital.

The one patient listed as being "questionable adenoma, questionable carcinoma" was presented in some detail in an earlier paper,<sup>9</sup> because the original diagnosis by the surgical pathologist was carcinoma. The patient was cured of a most marked hyperparathyroidism and is alive 21 years later without recurrence.

In only 3 patients were more than 1 adenoma removed, whereas the patient with carcinoma had had 2 previous operations at another hospital with the removal of 4 distinct tumors. In addition we removed 2 small isolated carcinomatous tumors from the cervical region and 2 from the lung. At autopsy a solitary metastatic tumor was found in the liver.

#### SUMMARY

Since hyperparathyroidism was first diagnosed and treated surgically in the United States 30 years ago, the medical profession has become much more aware of its early manifestations. Urinary calculi rather than skeletal changes are now more frequently the presenting finding that leads to the correct diagnosis. An elevated calcium and reduced phosphorus with calciuria are pathognomic of hyperparathyroidism. The differential diagnosis, therefore, is readily arrived at in patients with hypercalcemia suspected of having hyperparathyroidism.

Early surgical treatment interrupts the progress of the condition and results in reversal of many of the changes that have taken place. Where hyperparathyroidism has been long present even in a mild form certain conditions such as hypertension associated with deposition of calcium in the kidney is not reversible.

A greater awareness of the early clinical signs and symptoms, increased availability of facilities for establishment of the diagnosis, together with a better understanding of the technical problems encountered by the surgeon render hyperparathyroidism and its complications amenable to cure by surgical therapy.

#### APPENDIX

##### *Hyperparathyroidism*

##### Clinical Example

Patient: P.D.      Sex: F      Age: 20  
10 Hospital Admissions

First hospital: number of admissions, 6.  
Diagnosis: renal calculi (six admissions during time interval December 1, 1949 and August 10, 1950).

Second hospital: number of admissions, 2.  
Operations for renal calculi (surgery performed August 1950 and November, 1950).

Third hospital (The New York Hospital): number of admissions, 2. April 30, 1951, removal of parathyroid adenoma. Renal calculi present. November 29, 1951, removal of renal calculi.

Follow-up through April 27, 1957, no recurrence of renal calculi. Ca., 8.6.

Urinary calculi, particularly in young individuals, is an indication for complete investigation for hyperparathyroidism.

#### Differential Diagnosis

Other conditions associated with hypercalcemia

Renal rickets

Bone atrophy due to disease or disuse

Hypervitaminosis D

Milk alkali syndrome

Osteolytic metastatic carcinoma

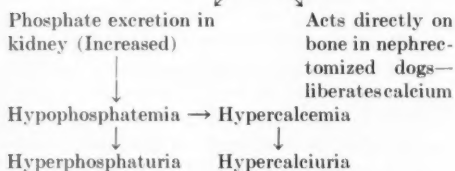
Multiple myeloma

Sarcoidosis (calcium values become normal with cortisone, ACTH)

#### Blood Determinations

	Hyperparathyroidism	Normal
Serum calcium	Normal to 22 mg. per cent	9-11 mg. per cent
Serum phosphorus	Lower than 2.9 mg. per cent	3-4.5 mg. per cent
Serum alkaline phosphatase	Normal to 30 u./100 mg.	1-4 u. (Bodansky method) 8-14 u./100 ml. (King Armstrong)

#### Excess of Parathyroid Hormone



#### Fate of Excess Calcium in Circulating Blood

Hypercalcemia

Nephrocalcinosis

Renal failure

Hypertension

Deposition in blood vessels throughout the body

Calcium loss in urine over 200 mg. (Low calcium diet)

Department of Surgery

The New York Hospital

New York 21, New York

#### REFERENCES

1. BURR, J. M., CARRELL, J. J., AND HILLS, A. G.: Sarcoidosis and hyperparathyroidism with hypercalcemia. *New England J. Med.*, 261: 1271, 1959.
2. CHURCHILL, E. D., AND COPE, O.: Parathyroid tumors associated with hyperparathyroidism. *Surg. Gynec. & Obst.*, 58: 255, 1934.
3. COFFEY, R. J., CANARY, J. J., AND DUMAIS, C. C.: Hyperparathyroidism and chronic calcific pancreatitis. *Am. Surgeon*, 25: 310, 1959.
4. COPE, O., KEYNES, W. M., ROTH, S. I., AND CASTLEMAN, B.: Primary chief-cell hyperplasia of the parathyroid glands: a new entity in the surgery of hyperparathyroidism. *Ann. Surg.*, 148: 375, 1958.
5. COPE, O., NARDI, G. L., AND CASTLEMAN, B.: Carcinoma of the parathyroid glands: four cases among 148 patients with hyperparathyroidism. *Ann. Surg.*, 138: 661, 1953.
6. ELKELES, A.: Parathyroid tumors with hyperparathyroidism and co-existent gastric and duodenal ulceration. *Lancet*, 1: 770, 1953.
7. ELLIS, J. T., AND BARR, D. P.: Metastasizing carcinoma of the parathyroid gland with osteitis fibrosa cystica and extensive calcinosis. *Am. J. Path.*, 27: 383, 1950.
8. FARRELL, J. J.: Physiologic consideration in the diagnosis and management of hyperparathyroidism. *Am. Surgeon* 25: 610, 1959.
9. GLENN, F.: Surgical treatment of hyperparathyroidism. *Ann. Surg.*, 149: 305, 1959.
10. HANNAN, R. R., SHORR, E., MCCLELLAN, W. S., AND DuBOIS, E. P.: A case of osteitis fibrosa cystica (osteomalacia?) with evidence of hyperactivity of the parathyroid bodies. *Metabolic study 1. J. Clin. Invest.*, 8: 215, 1930.
11. HELLESTRÖM, J.: Primary hyperparathyroidism. Observations in a series of 50 cases. *Acta endocrinol.*, 16: 30, 1954.
12. HOAR, C. S., JR., AND GORLIN, R.: Hyperparathyroidism and acute pancreatitis. *New England J. Med.*, 258: 1052, 1958.
13. KIRSNER, J. B.: Editorial. The parathyroids and peptic ulcer. *Gastroenterology*, 34: 145, 1958.

14. LACHER, M. J., GOLDBERG, J. A., THOMAS, D. F., AND CALVY, G. L.: Hyperparathyroidism and pancreatitis. Report of a case. *New England J. Med.*, 261: 239, 1959.
15. ST. GOAR, W. T.: Gastrointestinal symptoms as clues to diagnosis of primary hyperparathyroidism. Review of 45 cases. *Ann. Int. Med.*, 46: 102, 1957.
16. UNDERDAHL, L. O., WOOLNER, L. B., AND BLACK, B. M.: Multiple endocrine adenomas. Report of 8 cases in which parathyroids, pituitary and pancreatic islets were involved. *J. Clin. Endocrinol.*, 13: 20, 1953.
17. WERMER, P.: Genetic aspects of adenomatosis of endocrine glands. *Am. J. Med.*, 16: 363, 1954.

## MALIGNANT GRANULAR-CELL MYOBLASTOMA: REPORT OF A CASE AND REVIEW OF LITERATURE\*

D. T. HUNTER, JR., M.D., AND J. P. DEWAR, M.D.

*Oklahoma City, Oklahoma*

Although benign granular-cell myoblastomas occur frequently, the malignant variety is rare. Ross and associates stated that the reported frequency of malignant granular-cell myoblastoma seems to have been exaggerated by the inclusion of rhabdomyosarcomas and alveolar soft part tumors under this category.<sup>1</sup> In their review 4 authentic cases are cited, and 3 additional cases of malignant granular-cell myoblastoma are reported. Gamboa, in 1955, increased the number to 10 cases in his analysis of proven examples in the world literature.<sup>2</sup> He excluded 22 cases of alleged malignant granular-cell myoblastomas, because the photomicrographs and descriptions strongly resembled rhabdomyosarcomas, non-chromaffin paragangliomas, and alveolar soft part sarcomas. Gamboa also reported a case of malignant granular-cell myoblastoma of his own. Since 1955, 2 cases have been described, but the tumor described by Meredith and associates<sup>3</sup> must be excluded on the basis of the differentiation of malignant granular-cell myoblastoma from alveolar soft part sarcoma given by Christopherson and co-workers.<sup>4</sup> Meredith described the tumor as having a pseudoalveolar or organoid pattern and a fine granularity of the cytoplasm. Švejda and Horn<sup>5</sup> confirm Gamboa's analysis of the world literature and present a case that satisfies all of the criteria applied by Christopherson and others. To date there are 12 authentic cases of malignant granular-cell myoblastoma reported in the world literature. The case reported below is the 13th to fulfill the requirements generally accepted as characteristics of this neoplasm.

### CASE REPORT

A 73-year-old white woman was in good health until September 1957, when she began noticing generalized weakness and weight loss. In December 1957 she developed a sharp right lower quadrant pain, nausea with vomiting, fever, malaise and dizziness. She was hospitalized and had surgical removal of a ruptured gangrenous appendix. Her postoperative hospital course was

uneventful, but upon returning home she experienced episodes of burning, pressing "gas pains" in the epigastrium. Sharp right lower quadrant pains accompanied each attack of epigastric pain.

Careful evaluation of the patient after admission to the University of Oklahoma Hospitals revealed a chronically ill patient with a 23-pound weight loss, a sporadic low grade fever and generalized weakness during the preceding 11-month period. She had hyperactive bowel sounds, tenderness over the right abdomen, a palpable liver edge and a 5- by 8-cm. mass in the right lower quadrant. Her stools were black and Ham's test for occult blood in the stools was 4+. The hemoglobin was measured at 9.6 gm. per cent and the hematocrit at 29.0 per cent. The clinical impression was carcinoma of the colon.

The patient was explored and a tumor of the right colon was resected. No liver or lymphatic metastases were observed at surgery. The post-operative course was uneventful. Periodic examinations over a period of 15 months have failed to reveal recurrent tumor. Weight, hemoglobin, and subjective symptoms have remained stable. The liver, which was palpable before surgery, had regressed in size.

The surgical specimen (S-2919-58) consisting of 31 cm. of distal ileum and 23 cm. of right colon contained a 5 by 5 by 5 cm. mass in the large bowel approximately 4 cm. from the ileocecal valve. The mass completely circumscribed the large bowel and 3 loops of small intestine were attached to it. The tumor on section was grey and had firm consistency. Its margins were ill-defined. Except for one area of ulceration the mucosa was intact. The adhesions did not contain tumor.

Microscopic sections of the colon showed complete replacement of the muscularis by a poorly differentiated tumor (fig. 1). At the margin there was infiltration of normal tissue by tumor cells. The tumor had a medullary pattern and consisted of pleomorphic cells containing granular cytoplasm. Cell types varied from typical granular cells to highly anaplastic cells with less intense granulation (fig. 2). Mitotic figures were rare. Reticulum elements were prominent and wove indiscriminately around individual cells. No mucous was produced, nor were glands or other structures formed. Striations were absent. The lymph nodes

\* From the Department of Surgical Pathology, University Hospital, Oklahoma City, Oklahoma.



FIG. 1. Section of colon demonstrating intact mucosa, infiltrative character of tumor, and pleomorphism of cell population (hematoxylin and eosin,  $\times 100$ ).



FIG. 2. Section of tumor showing gradations of anaplasia. Some cells have small nuclei and large quantities of coarsely granular cytoplasm whereas other cells are very anaplastic. Distinct nucleoli are seen in many cells. Reticulum envelopes individual cells (hematoxylin and eosin, 450).

August

Diffe

Cell sh

Cytopl  
NucleuMitosi  
ReticuMicros  
of tu  
Struct

Locati

Growtl  
Metast

Special

were fr  
argyro  
Path  
myobla

The  
the ma  
ular-ce  
from p  
mor oc  
Gambo  
histolog  
is of th  
the 2 t  
cally be  
had a n  
logically  
of 52 y

The  
from al  
chroma  
rhabdo  
simple  
disorde

TABLE 1

*Differential characteristics of two tumors morphologically related to and frequently misdiagnosed as malignant granular-cell myoblastomas*

	Malignant Granular-Cell Myoblastoma (Histologically Malignant)	Alveolar Soft Part Sarcoma	Nonchromaffin Paraganglioma
Cell shape	Round to elongate	Large oval or polygonal	Round, fusiform, or stellate
Cytoplasm	Coarsely granular	Finely granular	Finely granular or clear
Nucleus	Small, fairly distinct nucleoli	Large, prominent nucleoli	Uniform, small, round vesicular or dense and pyknotic
Mitosis	Few	Many	None
Reticulum	Intercellular irregular pattern	Surrounds nests	Surrounds groups of cells
Microscopic appearance of tumor	Pleomorphic	Fairly uniform, organoid (Pseudoalveolar)	Uniform, medullary
Structure	Sheets, medullary	Nests separated by thin-walled vascular spaces	Thin-walled blood vessels in stroma between groups of cells
Location	Subcutaneous, soft tissue, smooth muscle	Striated muscle	Adjacent large vessels
Growth rate	Variable	Rapid	Slow
Metastases	Vascular and/or lymphatic	Vascular	None
Special stains	Argyrophilic (silver), granules orange-red (oil red "O")	Argyrophobic (silver), granules orange-red-purple (oil red "O")	Argyrophilic (silver)

were free of tumor. Special stains showed coarse argyrophilic granules in the cytoplasm.

*Pathologic diagnosis:* Malignant granular-cell myoblastoma, ascending colon.

The malignant granular-cell myoblastoma is the malignant form of the rather common granular-cell myoblastoma which is thought to arise from primitive neurogenous elements.<sup>6</sup> The tumor occurs in 2 distinct types referred to by Gamboa<sup>2</sup> and Howe<sup>3</sup> as histologically benign and histologically malignant. The case reported above is of the latter variety. The malignant behavior of the 2 types is identical. The 7 cases of histologically benign malignant granular-cell myoblastoma had a mean age of 32 years; 6 cases of the histologically malignant tumor had an age incidence of 52 years.

The tumor must be histologically differentiated from alveolar soft part sarcomas, malignant non-chromaffin paragangliomas, storage diseases, and rhabdomyosarcomas. In general this is relatively simple (table 1). Its differentiation from storage disorders and rhabdomyosarcomas offers no seri-

ous problem. Rarely the alveolar soft part sarcoma and the malignant granular-cell myoblastoma will have overlapping characteristics and special stains must be employed to enable accurate diagnosis. The alveolar or organoid pattern of some rhabdomyosarcomas may also offer problems of differentiation. Almost always striated cells are present to enable the diagnosis of rhabdomyosarcomas.<sup>7</sup> Location, special stains and malignant behavior will assist in differentiating malignant granular-cell myoblastomas from non-chromaffin paragangliomas.

Malignant granular-cell myoblastomas have occurred in adults between the ages of 21 and 73 years (table 2). Cases thus far described involve 10 women and 3 men. The mean age is 42 years, but no distribution curve can be constructed for a series of this size. Most of these tumors arise in the subcutaneous tissue of the head, neck and extremities. One case was reported in the bladder and another in the breast. Metastases are routed through both the lymphatics and blood stream seeding in the lungs, lymph nodes, liver, bone, ovaries, brain, spleen and body cavities. In sev-

TABLE 2

*Summary of cases of malignant granular-cell myoblastomas appearing in the literature*

Year	Author	Age	Sex	Histology	Location	Metastases	Treatment	Result
								mo.
1945	Ravich, Stout, and Ravich <sup>2</sup>	31	M	Benign, type I	Urinary bladder	Generalized	Excision, x-ray	Died, 17
1946	Powell <sup>2</sup>	26	F	Benign, type I	Subcutaneous, head and extremities	Ovaries, retroperitoneum	Excision	Alive, 11
1948	Dunnington <sup>2</sup>	40	M	Malignant, type II	Eyelid	Generalized	Excision	Alive, 24
1949	Ceelen <sup>2</sup>	45	F	Malignant, type II	Subcutaneous, arm	Lymphatics, breast and lung	Excision	Alive, 54
1951	Schwidde, <sup>2</sup> Meyers, and Sweeney <sup>2</sup>	21	F	Benign, type I	Subcutaneous, thigh	Cerebral, humerus	Excision, x-ray	Alive, 37
1952	Ross, Miller, and Foote <sup>1</sup>	60	M	Malignant, type II	Subcutaneous, back	Lymphatics, lungs	Excision	Alive, 36
1952		58	F	Malignant, type II	Subcutaneous, ankle	Lymphatics	Excision, x-ray	Died, 96
1952		33	F	Malignant, type II	Subcutaneous, thigh	Local	Excision	Alive, 8
1953	Crawford <sup>2</sup>	50	F	Benign, type I	Breast	Lung, liver and retroperitoneum	Symptomatic	Died, 12
1953		31	F	Benign, type I	Subcutaneous, trunk and extremities	Genital, mouth and neck	Excision, x-ray	Alive, 240
1955	Gamboa <sup>2</sup>	30	F	Benign, type I	Subcutaneous, thigh	Lymphatics, lung	Excision, x-ray	Alive, 60
1958	Svejda and Horn <sup>4</sup>	48	F	Benign, type I	Subcutaneous, groin	Generalized	Excision	Died, 36
1959	Hunter and Dewar	73	F	Malignant, type II	Colon	None demonstrated	Excision	Asymptomatic, 15

eral instances metastases did not appear until years after initial surgery.

Successful treatment entails complete removal of the primary lesion before metastasis. Subsequent surgery and x-ray therapy are felt to be of only palliative value. Although radiation sensitivity of the malignancy is not striking, 6 cases receiving radiation therapy survive 2 to 3 times longer than untreated cases. In those cases cul-

minating in death mean survival was 40 months after diagnosis.

## SUMMARY

A malignant granular-cell myoblastoma is described arising in the colon of a 73-year-old woman. This tumor is morphologically similar to alveolar soft part sarcomas and nonchromaffin

paragangliomas. A scheme for its differentiation is presented.

This sarcoma is very rare, and the case described is the thirteenth true malignant granular-cell myoblastoma to appear in the literature.

Dr. DeWitt T. Hunter

Baptist Memorial Hospital

5800 North West Grand Blvd.

Oklahoma City 12, Oklahoma

#### REFERENCES

1. ROSS, R. C., MILLER, T. R., AND FOOTE, F. W., JR.: Malignant granular-cell myoblastoma. *Cancer*, 5: 112, 1952.
2. GAMBOA, L. G.: Malignant granular-cell myoblastoma. *A. M. A. Arch. Path.* 60: 663, 1955.
3. MEREDITH, J. M., KAY, S., AND BOSHER, L. H., JR.: Case of granular-cell myoblastoma (organoid type) involving arm, lung, and brain with twenty years' survival. *J. Thoracic Surg.*, 35: 80, 1958.
4. CHRISTOPHERSON, W. M., FOOTE, F. W., JR., AND STEWART, F. W.: Alveolar soft part sarcomas: structurally characteristic tumors of uncertain histogenesis. *Cancer*, 5: 100, 1952.
5. ŠVEJDA, J., AND HORN, V.: Disseminated granular-cell pseudotumour. So-called metastasizing granular-cell myoblastoma. *J. Path. & Bact.*, 76: 343, 1958.
6. FUST, J. A., AND CUSTER, R. P.: On the neurogenesis of so-called granular cell myoblastoma. *Am. J. Clin. Path.*, 19: 522, 1949.
7. HORN, R. C., JR., AND ENTERLINE, H. T.: Rhabdomyosarcoma. A clinicopathological study and classification of 39 cases. *Cancer*, 11: 181, 1958.
8. HOWE, C. W., AND WARREN, S.: Myoblastoma. *Surgery*, 16: 319, 1944.

## THE SURGICAL TREATMENT OF SPASMODIC TORTICOLLIS\*

ALEXANDER T. BUNTS, M.D.

Cleveland, Ohio

In order to obtain optimal results from the surgical treatment of spasmodic torticollis, it is essential for the neurosurgeon to recognize the extremely diverse clinical variants of this disorder, so that he may select only those patients who are most likely to benefit from the procedure. In spasmodic torticollis the abnormal positions of the head may be due to tonic or clonic contractions of the neck musculature. The head may turn to one side only, it may bend to one side and rotate to the other, or it may incline backward (retrocollis) or forward (antecollis). A combination of these movements may occur; they may be rapid and jerking or deliberate and slow. In some cases the spasms may extend beyond the neck to involve the face, the shoulder and arm, and even other parts of the body, presenting the clinical picture of a more generalized dystonia. These spasms disappear in sleep, but are aggravated by emotional stress. In rare instances, the condition may have a psychogenic origin, although it is certainly difficult for the most careful observer to ascribe the violent, involuntary, and often uniformly patterned spasms of the cervical muscles to psychologic disorders such as nervousness, hysteria or psychoneurosis.

### SELECTION OF PATIENTS

If careful study and evaluation of a patient offers good reason to believe that the condition is primarily of psychologic or emotional origin, surgical treatment is contraindicated. In my experience the most satisfactory results of surgical treatment are to be expected in those patients who present a series of irregular, violent, jerky movements of the head, with rotation to one side only, and with the chin tilted upward. If the head is turned toward the left side, the right sternocleidomastoid muscle appears to be hypertrophied and to contract irregularly, and if turned toward the right side, the left sternocleidomastoid muscle appears to be affected. Other muscles,

such as the platysma, rectus capitis, obliquus capitis, trapezius, splenius capitis, semispinalis capitis, longus capitis and the scalenes are involved in the abnormal movements. These movements may begin gradually at any time of life, becoming more severe with the passage of years. The violent muscular contractions are not only uncomfortable, they may be painful, and cause considerable disability and actual physical interference with work.

A peculiar feature in some cases is that the patient is able to control the spasm by slight pressure against the chin, and to ward it off as long as the pressure is maintained. Surgery should be avoided in those cases in which spasmodic contractions of muscles are diffuse, involving shoulders, arms, back muscles and other parts of the body; such dystonias cannot be benefited by dividing nerves. It is not difficult to differentiate between spasmodic torticollis and the ischemic or traumatic wryneck of childhood which is caused by injury of the sternocleidomastoid muscle. In this type of wryneck there is a constant contraction that is due to scarring and infiltration of the muscle. Dysfunction of the extraocular muscles may result in the moderate head tilt of ocular torticollis.

### ETIOPATHOGENESIS

No single cause for spasmodic torticollis in man has ever been determined. The clinical manifestations of the condition suggest its possible relation to the dystonias and athetoses. Some years ago a few isolated autopsy studies<sup>2</sup> suggested the possibility that the disorder might have its origin in lesions of the corpus striatum of the brain, but firm proof of such a cause is lacking.

In 1952, Kemberling and associates<sup>3</sup> produced torticollis in cats by means of unilateral electrolytic lesions in the vestibular nuclei and reticular substance of the hindbrain. The result was evidently a sustained postural change of the head rather than a true spasmodic torticollis. In 1959, Foltz and co-workers<sup>7</sup> reported that they were able to induce true spasmodic torticollis in monkeys by producing electrolytic lesions in the medial

\* From the Department of Neurological Surgery, The Cleveland Clinic Foundation and The Frank E. Bunts Educational Institute, Cleveland, Ohio.

reticular formation of the midbrain. Kinnier Wilson<sup>10</sup> was of the opinion that the hysterical and occupational variants of torticollis probably originated in the cerebrum, whereas the spasmodic type seemed to have a spinobulbar origin. He suggested that the latter form might develop from the hysterical and occupational forms by a vicious circle of afferent impulses from a perpetually contracting muscle, whereby the movement, at first cortical, might become genuinely spasmodic.

#### TREATMENT

**Nonsurgical therapy.** Throughout the years, attempts have been made to relieve or to control spasmodic torticollis by many forms of nonsurgical or conservative therapy. Physical therapy, including the application of heat and massage to the neck, manipulation, head traction and electric treatments have been tried without success. Injections of procaine hydrochloride and alcohol in muscles have failed to bring relief. Braces and various types of collars have proved to be of little or no avail. Our experience with drug therapy, including sedatives, muscle relaxants, hyoscine, trihexyphenidyl (Artane, Lederle Laboratories) and cyerimine hydrochloride (Pagitane, Eli Lilly and Company) has been discouraging, but Foltz and associates<sup>7</sup> reported sustained objective improvement in 2 patients who received certain anticholinergic drugs. Psychotherapy has been reported to give favorable results in a few cases, but it seems most unlikely that such treatment can be expected to control the repeated violent contractions of chronic spasmodic torticollis.

**Surgical procedures.** Before 1891, surgical intervention to relieve the condition was limited chiefly to sections, resections, or tenotomies of the sternocleidomastoid muscle, although DeMorgan,<sup>4</sup> in 1866, was among the first to resect the spinal accessory nerve in the neck, thus paralyzing the muscle. Nerve-stretching was also tried without success. Finally it became apparent that paralysis of the sternocleidomastoid muscle alone failed to bring about satisfactory improvement and that the nerve supply to other rotator muscles of the neck must also be interrupted. In 1891, Keen<sup>8</sup> reported unilateral section of the posterior rami of the first 3 cervical nerves on the affected side at their exits from the intervertebral foramina.

Following a suggestion by Foerster<sup>6</sup> for reduc-

tion of incoming stimuli from the muscles in the relief of spasticity in Little's disease, Taylor<sup>13</sup> in 1915, reported the intraspinal attack upon spasmodic torticollis, performing unilateral section of the sensory nerve roots of the first 4 cervical nerves, together with section of the spinal accessory nerve in the neck. McKenzie<sup>11</sup> reported a case in which Cushing performed an intradural section of the spinal accessory nerve at the jugular foramen, and section of the motor and sensory roots of the upper 3 cervical nerve roots on the same side. Finney and Hughson<sup>5</sup> divided both spinal accessory nerves and the posterior rami of the upper 3 cervical nerves in the neck on both sides.

Dandy<sup>3</sup> reported 8 patients in whom he had performed intradural section of the motor and sensory nerve roots of the upper 3 cervical nerves on both sides, and bilateral section of the spinal accessory nerves; these patients were greatly improved. Later, in other patients he performed the same operation but preserved the posterior sensory nerve roots; the results were equally satisfactory. Since that time most neurosurgeons have used the latter operation for the relief of intractable spasmodic torticollis. Occasionally, division of the anterior nerve root of the 4th cervical nerve has been included in the procedure in order to produce more complete paralysis of the rotator muscles of the neck. In my experience, this nerve should be preserved, as its destruction increases the postoperative possibility of greater instability of posture of the head, and because of its contribution to the innervation of the diaphragm. In most patients the spasm of the sternocleidomastoid muscle is limited to only one side, and it is unnecessary to divide both spinal accessory nerves, since only the nerve that supplies the contracting sternocleidomastoid muscle is involved. Adson and associates<sup>1</sup> observed that Dandy's procedure sometimes resulted in subluxation and anterior curvature of the upper cervical vertebrae with forward falling of the patient's head, and interference with swallowing, breathing, and speaking. To avoid such a sequel they recommended the use of a bone graft extending from the occipital bone to the level of the 5th cervical lamina on both sides. This was performed immediately after completion of the nerve sections, and the head and neck were fixed in plaster for from 10 to 12 weeks. In our experience this procedure has not been necessary.

## DISCUSSION

Unsatisfactory results with conservative measures in the treatment of spasmodic torticollis, and the frequently favorable results with surgical treatment are the bases of my rather definite views regarding the management of the condition.

In the first place, if careful evaluation of the individual patient indicates that emotional or psychologic instability dominates the clinical picture, surgical treatment should not be recommended, and the aid of the psychiatrist should be sought first. In such cases, surgery, even though successful in paralyzing spastic muscles, almost never results in improvement of the patient's mental attitude toward the disorder. As stated previously, the patients with extensive dystonias that involve muscles outside of the neck are not suitable candidates for operation. When muscular spasms are limited to the neck and are severe enough to disable the patient from continuing his work, or when they cause great pain and constant discomfort to the patient, operation offers an excellent chance for improvement, if not cure. Occasionally the spasms may be of such a mild degree that the surgeon may consider surgery to be unjustified, and the patient may be able to adjust to the chronicity of the condition without excessive discomfort or unhappiness and may learn to live with it. On the other hand, in time, a mild form of torticollis may become severe and unbearable, and further consideration of surgical treatment may become advisable.

The operation that in my experience has given the most favorable results, includes intradural section of the anterior cervical nerve roots, and section of one spinal accessory nerve in the neck. With the patient in the sitting position, under thiopental sodium anesthesia, a complete laminectomy of the first 2 cervical vertebrae and removal of the upper part of the lamina of the 3rd cervical vertebra is performed; the dura is opened in the midline, and the anterior roots of the first 3 cervical nerves on both sides are divided between silver clips. Before dividing the nerve roots, the small arteries that accompany them should be teased away. Thus, important vessels supplying blood to the spinal cord are preserved, and possible neurologic deficit may be avoided.

Section of the posterior sensory nerve roots is not necessary to accomplish our purpose, and it causes loss of sensation in the occipital area of the

scalp and in the neck. Usually it is necessary to divide the spinal accessory nerve that supplies the sternocleidomastoid muscle involved in the spasmodic syndrome. This division can be performed about a week later, while the patient is still in the hospital, using an anterior approach in the neck. Some surgeons prefer to divide the spinal accessory nerve within the dura at the time of the cervical rhizotomy, but delay is not harmful, and in one instance it was found that section of the anterior cervical nerve roots alone sufficed to relieve the condition, and the spinal accessory nerve could be preserved, thus avoiding atrophy of the sternocleidomastoid and trapezius muscles.

To help the patient to arrive at a decision for or against surgical treatment, it is of the greatest importance to explain to him the operation, its purpose and its risks, and to evaluate its results. He should be told that no operation is completely satisfactory for this condition. In carefully selected patients, applying the operation as described above, one may anticipate almost complete cessation of the major violent jerking movements of the head. Minor fine tremors of the head sometimes persist postoperatively, but usually they are not too troublesome. Atrophy of the sternocleidomastoid and trapezius muscles always follows section of the spinal accessory nerve, and the resultant asymmetric appearance of the neck and posture of the head is of considerable concern to some patients, especially in the case of young women, to whom it must be thoroughly explained beforehand. There is usually some limitation of head movements after operation, especially in rotation of the head to the side opposite to that of the divided spinal accessory nerve.

After operation, dysphagia may be present for varying periods, but it usually subsides within a few weeks. Apparently the dysphagia is due to a flexed posture of the head, and the patient soon learns that he can swallow more easily when he exerts manual pressure against his forehead. Despite the occurrence of these minor sequelae, the relief of the former major, violent, spasmodic, jerking movements of the head permits the patient to relax and to obtain some rest; his morale and outlook for future comfort are improved; and his economic usefulness is enhanced. Poppen and Martinez-Niochet<sup>12</sup> followed 36 patients for from 1 to 7 years after operation for spasmodic torticollis and estimated that the results were satisfactory in 22 patients, and unsatisfactory in 14

patient  
the op  
agree  
and M  
were n  
head w  
Never  
to be  
sightly  
We ha  
we feel  
risk of  
Foltz  
the fut  
benefit  
rected  
in the  
ruption  
are dev  
advisa  
of the  
spinal  
spasmo

*The Cl  
Clevela*

1. An  
t  
C

patients. My own experience in 20 patients, with the operation as described above, leads me to agree completely with the statement of Poppen and Martinez-Niochet<sup>12</sup> to the effect that "none were normal in the sense that the attitude of the head was the same as before the disease started." Nevertheless, the majority of patients were happy to be free from their former disabling and unsightly torsion spasms of the cervical muscles. We had no operative mortality in 20 patients, so we feel justified in assuring the patient that the risk of death is not great.

Foltz and associates<sup>7</sup> have suggested that, in the future, spasmodic torticollis may possibly be benefited by stereotactic surgical procedures directed toward the central pathologic mechanisms in the brain stem, rather than by peripheral interruption of motor impulses. Until such procedures are developed and proved to be satisfactory, it is advisable to divide intradurally the motor roots of the first 3 cervical nerves on both sides and the spinal accessory nerve on one side, to relieve spasmodic torticollis.

The Cleveland Clinic Foundation  
Cleveland, Ohio

# REFERENCES

1. ADSON, A. W., YOUNG, H. H., AND GHORMLEY, R. K.: Spasmodic torticollis. Severe organic type treated by combined operation, rhizotomy, and fusion. *J. Bone & Joint Surg.*, 28: 299, 1946.
2. ALPERS, B. J., AND DRAYER, C. S.: Organic background of some cases of spasmodic torticollis; report of case with autopsy. *Am. J. M. Sc.*, 193: 378, 1937.
3. DANDY, W. E.: Operation for treatment of spasmodic torticollis. *Arch. Surg.*, 20: 1021, 1930.
4. DEMORGAN, C.: Case of excision of a part of the spinal accessory nerve for spasmodic wry neck. *Brit. and For. M.-Chir. Rev.*, 38: 218, 1866.
5. FINNEY, J. M. T., AND HUGHSON, W.: Spasmodic torticollis. *Ann. Surg.*, 81: 255, 1925.
6. FOERSTER, O.: On indications and results of excision of posterior spinal nerve roots in man. *Surg. Gynec. & Obst.*, 16: 463, 1913.
7. FOLTZ, E. L., KNOPP, L. M., AND WARD, A. A., JR.: Experimental spasmodic torticollis. *J. Neurosurg.*, 16: 55, 1959.
8. KEEN, W. W.: A new operation for spasmodic wry neck. Namely, division or excision of the nerves supplying the posterior rotator muscles of the head. *Ann. Surg.*, 13: 44, 1891.
9. KEMBERLING, S. R., BAIRD, H. W., AND SPIEGEL, E. A.: Experimental torticollis of rhombencephalic origin. *J. Neuropath. & Exper. Neurol.*, 11: 184, 1952.
10. WILSON, S. A. K.: *Neurology*, Vol. 3, p. 1961. Williams & Wilkins Company, Baltimore, 1955.
11. MCKENZIE, K. G.: Intrameningeal division of the spinal accessory and roots of the upper cervical nerves for the treatment of spasmodic torticollis. *Surg. Gynec. & Obst.*, 39: 5, 1924.
12. POPPEN, J. L., AND MARTINEZ-NIOCHET: Spasmodic torticollis. *S. Clin. North America*, 31: 883, 1951.
13. TAYLOR, A. S.: Operations on the peripheral and cranial nerves. Unilateral laminectomy. In: *Operative Therapeutics*, edited by A. B. Johnson, Vol. 1, Chap. 13, pp. 525-612. D. Appleton & Company, New York, 1915.

## RESTORATION OF FINGER FLEXION WITH HOMOLOGOUS COMPOSITE TISSUE TENDON GRAFTS\*

ERLE E. PEACOCK, JR., M.D.

*Chapel Hill, North Carolina*

Restoration of damaged flexor tendons within the digital sheath of human fingers is one of the most unpredictable procedures in reconstructive surgery. Perhaps no other restorative procedure requires so much attention to minute technical details for any chance of success, yet depends upon so many other factors which are not under the surgeon's control. The large number of technical stunts and mechanical gadgets which have appeared in past surgical literature attests to our frustration in attempting to control the special processes of wound healing which go on around a healing tendon or a free tendon graft. A majority of these technical innovations have been designed to prevent or reduce the formation of postoperative adhesions, which are essentially the end result of the collagenous or fibroprotein synthesis state of normal wound healing.

Ideally, the objective of reparative tendon surgery is to attain great tensile strength (coincident with new collagen production) between the ends of severed tendons or free grafts without developing fibrous adhesions between the tendon and its adjacent tissue. Such a goal is impossible to attain, however, because both the cells which produce the new collagen to amalgamate the ends of severed tendons as well as the blood supply which is necessary for the vitality of cells within the tendon grow into the tendon from surrounding tissues. Thus, healing between the tendon ends and revascularization of a free graft or damaged tendon to provide intrinsic nutrition is dependent upon connective tissue connections which contain a significant amount of fibrous tissue. In previously reported experiments, we have been able to show that successful isolation of the center of a tendon by mechanical devices which prevent connective tissue from forming between the tendon and its surrounding tissue will result in death of the cells of the tendon, disap-

pearance of ground substance, and failure of healing between the tendon ends.<sup>1-3</sup> In our opinion, the development of fibrous adhesions around healing tendons is a normal phase of wound healing which must occur around all tendons if they are to survive and maintain their normal physiologic functions. Just as surface scars usually soften and become more pliable (mature) with age, the fibrous tissue surrounding freshly repaired tendons usually develops some elasticity and eventually will permit the re-establishment of a satisfactory amplitude of motion between the tendon and its surrounding tissue. Just as some people apparently do not have the inherent capacity to prevent the formation of excess collagen and develop elasticity in surface scars, there are some individuals who do not seem to be able to develop elasticity in deep scar tissue, thus accounting for the failure of some tendon grafts to glide, regardless of the skill of the operator or the integrity of the patient. Such patients may be comparable to people who form hypertrophic scars or keloids following surface restorations.

Because we are not presently able predictably to control the physical characteristics of new fibrous tissue in healing wounds, it has seemed that our best opportunity to prevent the restricting features of new fibrous tissue, without interfering with the blood supply to healing tendons, would be to search for a method of tendon grafting which would cause postoperative adhesions to develop in an area where they would not interfere with motion, but would still permit revascularization of the graft. With this objective in mind, we have been experimenting with a tendon preparation which we have called a composite tissue tendon graft. The graft consists of two flexor tendons with an intact fibrous sheath. The specialized blood vessels which course through the vincula and volar mesentery are an important part of the graft, for they connect the periosteum or phalangeal surface of the sheath with the enclosed tendons. Theoretically, the normal healing process, including the production of fibrous tissue, would occur around the exterior of the fibrous

\* From the Department of Surgery, University of North Carolina School of Medicine, and the North Carolina Memorial Hospital, Chapel Hill, North Carolina. Supported in part by Grant No. A-2151 from the United States Public Health Service.

sheath  
within  
vessels  
nourish  
fer. Fi  
the ex  
ternal  
would  
being  
depict  
moved  
A m  
have b  
plante  
rabbit  
mals f  
rangin  
were s  
be str  
geneti  
solidly  
the an  
was in  
bathed  
there



FIG. 1. Dissection of cadaver hand to remove a composite tissue tendon graft

sheath; thus the sheath would be revascularized within a few days. The specialized elastic blood vessels in the vincula and mesentery could then nourish the tendon as they did before the transfer. Fibrous adhesions would develop only around the exterior surface of the sheath, and the internal gliding surface between tendons and sheath would be preserved. Figure 1 shows such a graft being removed from a human finger, and figure 2 depicts the graft after it has been completely removed.

A number of composite tissue tendon grafts have been excised from human fingers and transplanted beneath the abdominal wall of dogs and rabbits. These grafts were removed from the animals for gross and histologic study after periods ranging from 2 months to 1 year. Twelve grafts were studied in this manner and all were found to be structurally intact following their domain in a genetically different host. All of the grafts were solidly attached to the surrounding soft tissue of the animal's abdominal wall. When the sheath was incised, the flexor tendons were found to be bathed in normal-appearing synovial fluid, and there were no adhesions between the internal sur-

face of the sheath and the external surfaces of the enclosed tendons. The blood vessels coursing through the mesentery and vincula were functioning as evidenced by their ability to transport  $P^{32}$  from the donor animal's general circulation to the interior of the transplanted tendons. Figure 3 reveals the appearance of a dissected graft 6 months following transplantation beneath the abdominal wall of an adult mongrel dog.

The specialized relations of 2 tendons with a mesentery and vincula completely enclosed by a fibrous sheath is found only in the upper extremity of human beings and monkeys. The small size of composite tissue grafts removed from a monkey's hand eliminates the possibility of utilizing a zoograft to reconstruct human fingers; therefore, all of our grafts were taken from human cadavers shortly after death. Because there would seldom be any advantage in robbing one finger to restore another one, composite tissue tendon grafts of the flexor mechanism must be successfully transplanted as homografts if they are to be of any clinical value in the restoration of flexor tendon injuries.

The clinical application of tendon homografts



FIG. 2. Composite tissue tendon graft (includes flexor sublimis and flexor profundus tendons within an intact digital sheath).

to the problem of reconstructive surgery has raised several questions, such as the length of time that may elapse following death and removal of a graft, the method and length of time that a graft can be safely stored, the antigenicity of tendon, and numerous technical details concerning the length of time that immobilization is needed during the healing process. The results of a series of basic experiments which were performed to help answer some of these questions have been published in more detail in previous papers.<sup>4-6</sup>

A composite tissue tendon graft is essentially a nonliving, primarily collagenous, homostatic



FIG. 3. Dissection of a human composite tissue tendon graft 6 months after transplantation into the abdominal wall of a dog.

graft. The donor fibrocytes and ground substance are completely removed by the host animal, after which, the graft is invaded by host monocytes, many of which metamorphose into mature longitudinally oriented fibrocytes. During the 6th through the 15th day after transplantation, when the tendon is relatively acellular and without demonstrable interfibrillar ground substance, it has no lateral tensile strength and healing between the ends does not occur. After recellularization, ground substance re-appears and the tendon regains lateral stability. Healing between the ends of autogenous and homologous tendons requires at least 4 weeks. We have had one composite tissue tendon graft break at the point of its anastomosis with the host's tendons shortly after motion was started at the end of the 3rd postoperative week.

Because composite tissue tendon grafts are es-

entially the con-  
transfer  
have co  
there do  
host re  
ferred i  
which h  
and ant  
ments w  
genicity  
genicity  
animal  
could be  
Thus, ev  
to recei  
pletely  
to recei  
perimen  
grafts w

In ou  
obtain p  
person t  
instance  
tendon  
autopsy  
the age  
we have  
of neopl  
excised  
sequent  
with no  
and ove  
fore, the  
the sam  
course,  
finger as  
should  
the left  
on displ

The h  
the han  
gical op  
of the f  
midlater  
of the fl  
anism sh  
tissue fl  
palm an  
by mak  
the late

essentially nonliving grafts, we do not believe that the condition of the fibrocytes at the time of transfer is of any importance. The grafts do not have to be removed rapidly after death, and there does not appear to be any difference in the host reaction to grafts which have been transferred immediately and the reaction to grafts which have been stored in buffered saline solution and antibiotics for 40 days. A series of experiments which were designed to evaluate the antigenicity of tendon did not reveal sufficient antigenicity to cause a second set rejection in the host animal or a collagen antibody response which could be measured by complement fixation tests. Thus, even though preparation of a human finger to receive a composite tissue homograft completely prevents the finger from ever being able to receive a subsequent autograft, our basic experiments indicated that a clinical trial of these grafts was justified.

#### TAKING THE GRAFT

In our experience, it has not been difficult to obtain permission from the family of a deceased person to remove a flexor tendon graft. In several instances we have received permission to take a tendon graft when permission to perform an autopsy was denied. We have not thought that the age of the donor was a restricting factor, but we have not taken grafts from people who died of neoplastic or infectious diseases. One graft was excised from a jaundiced individual and was subsequently transplanted into another human being with no recognizable adverse effects. The length and over-all size of the graft is important; therefore, the size of the hand should be approximately the same size as the recipient's hand and, of course, the graft should be taken from the same finger as the damaged recipient finger. The graft should be removed from the right hand because the left hand is usually exposed when the body is on display.

The body is taken to the operating room and the hand and finger nails are prepared for a surgical operation. A volar incision down the center of the finger will be less visible than the usual midlateral incision and will afford better exposure of the flexor mechanism. The entire flexor mechanism should be exposed by reflecting lateral soft tissue flaps and by extending the incision into the palm and wrist. Dissection of the graft is started by making sharp incisions in the periosteum on the lateral surfaces of the phalanges. Sharp dis-

section in a subperiosteal plane is utilized to elevate the entire flexor mechanism from the phalanges. Blunt dissection with a periosteal elevator will often result in tearing or opening the sheath at the point of insertion of the sublimis slips to the middle phalanx or insertion of the profundus tendon into the distal phalanx. The sharp edge of the knife blade should be kept firmly against the bone as these places are dissected. Between the heavy condensations of fascia which serve as pulleys, the dorsal and volar surfaces of the sheath may be extremely thin; a tiny rent in the sheath will occasionally be produced in these areas. This does not ruin the graft for clinical use; we have merely closed the rent with a few carefully placed sutures before continuing the dissection. The interphalangeal and metacarpal phalangeal joints cause some difficulty because of the undulating surface of the flexor tendons in these areas. A careful review of the interdigital course of the tendons is valuable in that it helps the operator to anticipate sudden variations in the direction of the tendons before the sheath is inadvertently entered. The volar plate and capsule of the joints are too adherent to the dorsal layer of the tendon sheath to be stripped away, and so we have left these structures attached to the graft. Once the proximal palmar crease has been reached, the entire sheath has been salvaged, and the remainder of the tendons can be excised easily. The lumbrical muscle should be cleanly dissected away from the profundus tendon, and the volar carpal ligament should be split so that the tendons can be lifted out of the wrist without excessive traction. We have found that grafts which extend into the wrist will function better than those in which the proximal suture line is placed in the palm; therefore, we always take the full length of both flexor tendons in the forearm.

The excised graft is carefully inspected for any small rents in the sheath; these should be repaired with closely spaced fine sutures to produce a water-tight closure. The graft is then wrapped in a gauze sponge which is damp with physiologic saline to which aqueous penicillin and streptomycin have been added. The sponge surrounding the graft is placed in a small screw-cap specimen bottle and refrigerated at 37°F. until the recipient finger has been prepared.

After removal of the graft, the divided ends of the volar carpal vascular arches should be identified and doubly ligated. Any other significant vessels which have been divided during the dis-

section of the palm or wrist should also be ligated. An extremely annoying leak of embalming fluid will occur for several days if this important maneuver is neglected. The skin flaps should be accurately approximated with a tight, closely spaced, continuous suture. A No. 40-wire running suture has been very satisfactory and is scarcely noticeable even if the hand is exposed.

#### TRANSPLANTATION OF THE GRAFT

The damaged finger is prepared to receive the graft by opening it along the midlateral line. The incision should curve into the palm and extend proximalward to the proximal volar skin crease. After the skin flaps have been reflected, the flexor tendons are stripped out of the palm and finger, and all of the digital sheath is excised except for three or four tiny periosteal tags which are preserved at several points along each side of the finger. These tags will be used later as anchoring points to hold the transplanted sheath in proper alignment. A tag of periosteum is also turned up in the center of the distal phalanx to suture the insertion of the profundus tendon and tip of the transplanted sheath to the bone. The volar plates overlying the interphalangeal and metacarpal phalangeal joints are preserved to avoid opening the joint capsules. The lumbrical muscle is detached from the flexor profundus tendon in the palm and both flexor tendons are then withdrawn through a separate incision in the wrist.

The graft is placed in the finger and sutured to the periosteal tags along the sides of the phalanges and to the tip of the terminal phalanx. The tendons are threaded through the carpal canal and pulled out through the wrist incision. The finger and palm incisions should be closed before the proximal tendon anastomoses are performed. Proper tension on the index finger is adjusted by allowing the anesthetized hand to assume a neutral position and then applying tension on the proximal ends of the tendon grafts until the reconstructed finger comes into the same attitude of flexion as the normal fingers. It is advisable to shorten the tendons an additional few millimeters so that the reconstructed finger is slightly more flexed than the normal ones. There is a tendency for homografts to lengthen a little bit during their revascularization and recellularization phases; therefore, we have made homografts slightly shorter initially than we make conventional autografts. The tendons are joined to the proximal

host tendons by using a buried braided wire suture. We have repaired both sublimis and profundus tendons, but we have been careful to stagger the level of the suture lines. Postoperatively, we have not been able to separate sublimis and profundus function in our patients; but we have thought that the additional effect of the powerful, independently acting, sublimis tendon was beneficial in helping to overcome the restricting effect of postoperative adhesions around the anastomosis sites and the exposed tendons between the wrist and the beginning of the digital sheath in the palm (figs. 4 through 7).

After closure of the wrist incision, the hand is immobilized in a bulky occlusion dressing with the wrist flexed approximately 25 degrees to relieve tension on the proximal tendon suture lines. We remove the dressing at weekly intervals to take out cutaneous sutures and to put the interphalangeal joints through a normal range of passive motion. We have not permitted active use of the digits for 30 days because of the danger of dehiscence of the proximal suture line.

The only difference which we have observed in the immediate postoperative appearance of hands which have received homologous tendon grafts when compared with hands which have received autogenous grafts is that there is usually a little more edema which lasts a little longer in the hand which has been homografted. For this reason we have left some of the skin sutures in several days longer than usual.

#### RESULTS

Recovery of active flexion in fingers which have received composite tissue tendon homografts is very similar to the recovery of motion in fingers which have been reconstructed with conventional autografts. Only a flicker of terminal flexion is present at the first examination, and improvement may be observed for as long as 2 months after the beginning of exercises and physiotherapy. Development of motion is dependent upon the attenuation and relaxation of fibrous adhesions around the exposed tendons in the wrist and palm, and around the anastomoses sites in the wrist. The development of some elasticity in these adhesions, although considerably more certain in the wrist and palm than the development of elasticity in adhesions in the distal pulley system surrounding autografts,

FIG. 4.  
rated flexor  
carpoph

FIG.  
4 to sh  
graft in  
involv  
that o  
Fou  
transp



FIG. 4. Preoperative view of finger with lacerated flexor tendons. No flexion distal to metacarpophalangeal joint.



FIG. 6. Postoperative view of hand shown in figure 4.



FIG. 5. Operative view of hand shown in figure 4 to show extension of composite tissue tendon graft into the wrist.



FIG. 7. Functional postoperative view of hand shown in figure 4. Note normal flexion of the long finger.

involves the same basic alteration of collagen that occurs after an autograft.

Four composite tissue tendon grafts have been transplanted to human fingers. Table 1 sum-

marizes the indications for the operations and the results to date from these procedures. The results of 2 grafts have been classified as excellent because a full range of active motion was restored.

TABLE 1  
*Homologous composite tissue tendon grafts in human fingers*

Patient	Injury	Immediate Care	Time between Injury and Composite Graft	Passive Motion Preoperatively	Passive Motion Restored to Active Motion	Result
LSM	Multiple lacerations of both tendons, index finger at level of proximal phalanx	Primary suture attempted. Complicated by tenosynovitis	37. 2½	% 50	% 100	Excellent, 23 months
JWG	Laceration both tendons at level of proximal phalanx, long finger	Primary repair attempted; no function	¼	75	30	Poor, finger amputated
DG	Laceration both tendons at level of proximal phalanx, long finger during early childhood	Skin sutured; no tendon repair	22	100	100	Excellent, 20 months
CH	Laceration both tendons at level of proximal phalanx, fifth finger	Primary repair attempted	1	75	50	Good at 3 weeks; did not return after first post-operative examination

In 1 case, the interphalangeal joints were normal and flexion of the finger tips into the palm was possible. The other had severely damaged interphalangeal joints which allowed passive flexion to within only 1 cm. of the palm; because the composite tissue homografts completely restored active motion to the full limits of passive motion, this case has also been classified as an excellent functional results. A 3rd case recovered 50 per cent flexion by the end of 3 weeks which is an excellent early result. Unfortunately, this patient left the country rather abruptly, and we have been unable to contact him since the 3-week examination.

One graft functioned very poorly; only 20-degree active flexion was possible in the proximal interphalangeal joint, and there was no active flexion in the distal interphalangeal joint. Passive motion in these joints was almost normal. The finger containing this graft was amputated at a later date. Dissection of the finger revealed the gliding mechanism within the transplanted sheath to be intact, but satisfactory flexion of the finger did not develop because of extensive fibrous adhesions which were found between the host and the exposed tendons at the level of the proximal anastomoses in the wrist. Adhesions

develop around all grafts at this point, but, because many of the tendons pull together normally in the wrist, adhesions at this level are usually not incapacitating. Whether the unusually dense, unyielding adhesions which developed in this patient were due to a subclinical infection, developed secondary to a severe local foreign tissue inflammatory reaction, or were the result of some peculiar healing reaction in the individual, is unknown. A similar type of dense unyielding cicatrix spoils the functional result in many patients after restoration of flexor tendons by autografts, and so it is possible that this type of response in 1 of the 4 patients receiving composite tissue homografts was not related to the transfer of genetically foreign tissue.

#### SUMMARY

Three composite tissue homografts of the entire flexor mechanism have been observed to function satisfactorily in human fingers. A fourth graft did not function satisfactorily but was structurally intact after one year. Preliminary observations on the fate of composite tissue heterografts in laboratory animals indicates that such grafts persist primarily as collagenous structures until the donor cells and ground sub-

stance ha-  
tion and  
immuniz-  
from the  
foreign co-  
collagen  
fixation t-  
antigen.  
use of  
grafts ha-  
flexor ter-  
sheath an-  
utilized t-  
successfu-  
was possi-  
North Ca-  
Chapel H-

stance have been replaced by host cells. Destruction and replacement of the donor cells does not immunize animals against subsequent skin grafts from the same donor animal, and the presence of foreign collagen does not produce a rising titer of collagen antibodies as measured by complement fixation test utilizing reconstituted collagen as an antigen. At this time, clinical indications for the use of homologous composite tissue tendon grafts have been limited to severe injuries of the flexor tendons within and including the digital sheath and overlying soft tissues. We have not utilized the graft in less complicated cases where successful repair by a conventional autograft was possible.

*North Carolina Memorial Hospital  
Chapel Hill, North Carolina*

## REFERENCES

1. PEACOCK, E. E., JR.: The vascular basis for tendon repair. In *Surgical Forum*, Vol. 8, p. 65. American College of Surgeons, Chicago, 1957.
2. PEACOCK, E. E., JR.: A study of the circulation in normal tendons and healing grafts. *Ann. Surg.*, 149: 415, 1959.
3. PEACOCK, E. E., JR.: Some problems in flexor tendon healing. *Surgery*, 45: 415, 1959.
4. PEACOCK, E. E., JR., AND PETTY, J. M.: Biological reactions to collagen transplants. In *Surgical Forum*, Vol. 9, p. 815. American College of Surgeons, Chicago, 1958.
5. PEACOCK, E. E., JR., AND PETTY, J. M.: Antigenicity of tendon. *Surg. Gynec. & Obst.*, in press.
6. PEACOCK, E. E., JR.: Morphology of homologous and heterologous tendon grafts. *Surg. Gynec. & Obst.*, 109: 735, 1959.

## SURGICAL AXIOMS

EDWARD F. SKINNER, M.D.\*

Memphis, Tennessee

Occasionally we become so engrossed in operative technique that we may forget surgical fundamentals.

1. *The simple operation is the best operation.* The more steps and anastomoses in an operative technique, the more chance there is for complications.

2. *The patient's welfare is always the prime consideration.* Training of residents and internes is necessary but is a subordinate consideration.

3. *Fingernails must be trimmed very short.* This applies to the surgeon and his assistants, and to the scrub nurse. At the conclusion of an operation, about 25 per cent of the rubber gloves have holes in them. By the end of the operation, about 90 per cent of the cultures from the hands of the operating team show a staphylococci growth.

4. *Whenever possible, avoid implanting foreign material in the wound.* Foreign objects in a wound will frequently be returned to the surgeon later, like bread cast upon the water.

5. *The loquacious surgeon is blessed with more than his share of postoperative wound infections.* The brilliant conversationalist has no place in an operating room; no mask is completely impenetrable to mouth and nose organisms.

Be careful in dictating operative reports; your remarks may be read back to you later on in

court. "Inadvertent" means "careless." Never use it in dictating operations for obvious medical reasons. Dictate mishaps as "accidental" instead.

6. *A wet drape or gown is a contaminated linen.* Replace it.

7. *Proper exposure and light are necessary to good surgery.* Here an alert assistant surgeon is invaluable, along with a circulating nurse tall enough to reach the lights.

8. *Cardiac arrest* requires one instrument only—a knife—plus mouth-to-mouth breathing until an anesthetist is available to help. Electrical stimulators and drugs come later.

9. *Gentleness in handling tissue is more blessed than speed.* Avoid crushing clamps on any tissue which is to remain in the patient. A job done right today will not be an emergency tonight. Tie all sutures carefully, with three throws on important sutures.

10. *Keep the patient alive* (the last is the greatest commandment) even if it means a poor cosmetic result, or the loss of an extremity. You can always reoperate at a later day. Interrupt or stage the operation if necessary. Know before surgery at which point the operation can be stopped, and where the point of no return is reached.

Baptist Medical Building  
20 South Dudley Street  
Memphis 3, Tennessee

\* From the University of Tennessee College of Medicine, Department of Surgery, Memphis, Tennessee.

Pancre  
mortality  
disease is  
drink hea  
onset occ  
explained  
active at  
small hem  
the action  
frequently  
cholelithi  
of Vater  
back into  
Acute p  
gravity,  
festations  
orrhage w  
puration.  
combined  
or suppu  
considere  
hemorrha  
condition  
escaping  
pancreas  
pancreati  
added, s  
fulminati  
one of th  
with unus  
be difficu  
Subacu  
milder de  
present in  
Chromi  
from the  
stones, in  
or in the  
commone  
extension  
duodenal  
denal ulc  
duct; pos  
and alcoh  
Postop  
usually

## PANCREATITIS

LEONARD A. BIBLE, M.D.

*Jackson, Mississippi*

Pancreatitis is a serious disease with a high mortality regardless of the plan of treatment. The disease is more common in men who eat and drink heavily. The fact that in many patients the onset occurs 1 or 2 hours after a heavy meal is explained by recalling that the pancreas is most active at that time, and that any injury, such as a small hemorrhage or distention of a duct, favors the action of trypsin and autolysis. The disease is frequently associated with cholecystitis and cholelithiasis. A calculus lodged in the ampulla of Vater may dam up the bile until it is forced back into the pancreatic duct.

*Acute pancreatitis* is an involvement of extreme gravity, often characterized among other manifestations, by acute inflammatory changes, hemorrhage within and outside of the gland, and suppuration. These conditions may occur variously combined; inflammation, hemorrhage, gangrene or suppuration. Hemorrhagic apoplexy has been considered a condition of acute aseptic pancreatic hemorrhage; in contradistinction to the same condition associated with sepsis. In addition, the escaping pancreatic ferments, from the portion of pancreas involved cause fat necrosis, and peripancreatic peritonitis and when bacteria are added, suppuration. The condition may be fulminating and whether so or not, may prove one of the most unmanageable surgical problems with unusually high mortality. The diagnosis may be difficult or impossible.

*Subacute pancreatitis* has, essentially, but to a milder degree, the same characteristics which are present in the acute form.

*Chronic pancreatitis* is a condition which results from the following causes: irritation from gallstones, impacted either in the ampulla of Vater, or in the lower end of the common bile duct (the commonest cause); from pancreatic calculi; from extension of inflammation originating in gastroduodenal catarrh; extension from gastric or duodenal ulcer; outside pressure; obstruction of the duct; possibly from injuries and typhoid, syphilis and alcohol.

*Postoperative pancreatitis* is a condition which usually occurs after common duct surgery,

gastrectomy or splenectomy, and manifests itself by acute tachycardia, distention, prolonged illness or unexplained upper abdominal pain and tenderness. The diagnostic features are essentially those of acute pancreatitis including elevated serum amylase.

*Traumatic pancreatitis*, usually results from trauma, an example being the so called "steering wheel injury." The serum amylase is usually elevated, and the disease may be complicated by a high incidence of pancreatic pseudocyst.

The morbidity of this disease has been pointed out and the arbitrary divisions, depending upon the severity, have been listed. Accurate diagnosis of the disease, pancreatitis, presents a problem to tax the diagnostic acumen of the attending physician since there are no clear-cut symptoms or signs. In the acute hemorrhagic and suppurative types the onset is characterized by sudden constant agonizing epigastric or periumbilical pain, nausea, vomiting and symptoms of collapse. Rapid pulse and cyanosis are not uncommon. There is usually extreme tenderness of the pancreatic area extending into the costovertebral angles. However, these same signs and symptoms are too often found in the acute coronary occlusion, acute intestinal obstruction, acute gall bladder disease and especially peptic ulcers. There are a few features which may be utilized as an aid in establishing a proper diagnosis, such as close cardiac examination, abdominal percussion to determine the absence or presence of free air and to determine the absence or presence of liver dullness. Placing the patient in a left lateral position followed by x-rays to determine free air, is of value. The determination of serum amylase is most important. The serum amylase will be elevated in pancreatitis often enough to be used as the chief diagnostic aid. The findings of an elevated serum amylase in the presence of acute upper abdominal pain with tenderness across the upper abdomen points very emphatically to the diagnosis of acute pancreatitis.

In chronic pancreatitis we are faced with greater difficulties in establishing the correct diagnosis. Chronic pancreatitis may be confused

with milder forms of cardiac, cholecystic and gastrointestinal disease. It may actually never produce enough symptoms to make any diagnosis other than one labeled "gastritis", "indigestion" or some other vague syndrome. The less severe forms of pancreatitis do not produce shock, rapid pulse, cyanosis, etc., as in the severe forms. Should we, seek some aberration in glucose tolerance or serum amylase, we then might establish an accurate diagnosis, but all too often our patient is subjected to diagnostic exploratory laparotomy, and only then does the nature of the patients illness become manifest.

#### TREATMENT

##### *Surgical*

Surgical treatment may be summed up by saying that there is no adequate surgical treatment for acute pancreatitis. To review surgical history, the original idea was to incise the pancreatic capsule and drain the pancreas. The majority of the patients expired. The pendulum then swung toward no surgical intervention, but rather to treat the patient purely from a medical standpoint. It became popular to perform cholecystostomy, the theory being that pancreatitis usually occurs when there is some obstruction in the region of the ampulla of Vater. Such obstruction may result from inflammatory lesions of the duodenum, stones in the ampulla of Vater or some other related obstruction such as lesions in the head of the pancreas. When obstruction occurs, it is believed that a reflux of bile is forced into the pancreas and the syndrome of pancreatitis results. Consequently, by doing cholecystostomy, an "escape valve" is produced, and the pressure created in the biliary system is relieved through the drainage tube placed in the gall-bladder. This theory sounds plausible. It probably is of value when the obstruction is of the type just mentioned. It is useless in a diffuse pancreatitis not due to an obstructive etiology or a pancreatitis developed in a gland proximal to a pancreatic calculus. If a diffuse pancreatitis exists surgery will benefit only those who are obstructed within the duodenum or ampulla of Vater or head of the pancreas.

##### *Sphincterotomy*

The success of this operation has varied in the hands of different surgeons. Some have reported

very satisfactory results, others are less enthusiastic. One is justified in trying this procedure before attempting more radical surgery if no definite cause of disease is apparent on abdominal exploration.

##### *Jaundice*

Jaundice produced by pancreatitis in the presence of an undiseased gall bladder should be treated by some form of biliary tract drainage. As the common duct should always be explored, external drainage can readily be instituted by the use of a T-tube. Internal drainage may be established when obstruction to the flow of bile into the duodenum exists. If the cystic duct is patent, anastomosis between the gall bladder and some portion of the gastrointestinal tract will establish adequate internal drainage. Cholecystogastrotomy is a simple and usually satisfactory procedure.

The gall bladder may be anastomosed to the duodenum or to the jejunum, depending upon the ease of approximation and the preference of the surgeon.

When the cystic duct is obstructed or the gall bladder has been removed or is diseased, the common duct may be anastomosed to the duodenum or jejunum. A side-to-side anastomosis may be performed between the common duct and the duodenum. Some surgeons prefer to divide the common duct and anastomose its proximal end to the duodenum or jejunum.

##### *Pancreatic Cysts*

Symptomatic cysts should be treated surgically. There are 2 primary objectives to be derived from surgical treatment, either to remove the cyst or to establish drainage. Drainage may be of the "internal or external" type, that is to say marsupialization or anastomosis between the cyst and jejunum. Obviously complete eradication of the cyst is preferable, but all too often this cannot be done and the other types of treatment must be instituted. Generally a pancreatic cystojejunostomy form of internal drainage can be done and certainly carries a lower mortality and morbidity rate. Location, size, accessibility and point of origin will determine the type of procedure.

##### *Medical*

After the swing of the pendulum over the past several years, it is now generally accepted that

treatment  
not a su  
conserva  
portant  
and onc  
initiated

1. Rec  
complish  
mouth,  
adminis

2. Ele  
and nor

3. Th  
large tra  
common  
averagin  
much m

4. It  
should b  
sphincte

5. Sp

6. A  
adminis  
given be

It is l  
acute p

treatment of acute pancreatitis is a medical and not a surgical problem. After this trend toward conservative management it is obviously important that the diagnosis must be established and once the diagnosis is established, therapy initiated.

1. Reduction of pancreatic secretion is accomplished by giving the patient nothing by mouth, the use of nasogastric suction, and the administration of some type of vagolytic drug.

2. Electrolyte depletion should be overcome and normal fluid balance maintained.

3. These patients usually require frequent large transfusions of whole blood. It is not uncommon to find an initial blood volume deficit averaging 1500 cc. Usually the patient requires much more during the course of his illness.

4. It is necessary to relieve pain, but morphine should be avoided since it produces spasm of the sphincter of Oddi.

5. Splanchnic blocks may be considered.

6. A broad spectrum antibiotic should be administered and generally a tetracycline is given because this drug is excreted in the bile.

It is believed advisable to avoid surgery in the acute phase. After the patient has improved

under these therapeutic suggestions, he is maintained on vagolytic drugs and a low fat bland diet. Approximately 6 to 8 weeks after the acute phase of the illness a cholecystogram and upper gastrointestinal series should be obtained. Approximately 50 per cent of the patients will have biliary disease, and occasionally a pancreatic cyst will be found. In patients who exhibit biliary disease appropriate surgery should be considered.

Approximately 25 per cent of all patients who recover will have recurrent attacks, either of the acute or chronic type.

#### SUMMARY

Pancreatitis, in its various forms, is a disease of which we have little understanding as to etiology, physiology, pathology and treatment.

Medical therapy is preferable to surgery, and will generally afford better results as well as a greater survival ratio.

The indications for surgery are noted.

1151 North State Street  
Jackson 2, Mississippi

## Book Review

---

*The editors of THE AMERICAN SURGEON will at all times welcome new books in the field of surgery and will acknowledge their receipt in these pages. The editors do not, however, agree to review all books that have been submitted without solicitation.*

---

*Combined Textbook of Obstetrics and Gynaecology.*  
Edited by DUGALD BAIRD. Ed. 6, 936 pp., Williams & Wilkins Company, Baltimore, 1957. \$15.00

Since 1923, this has been a standard text for students and practitioners in Great Britain. Seventeen authorities, including such distinguished men as James Young, James Walker, W. I. C. Morris, and the editor, Dugald Baird, have contributed to the present edition.

In several respects there are some differences to be found between this volume and the standard American text. For instance, in a short discussion of obstetric anesthesia and analgesia, much

emphasis is laid on chloroform as a reasonable safe analgesic as well as an anesthetic agent. In addition, the use of nitrous oxide and oxygen is thoroughly approved of for relief of first stage labor pain. Neither of these agents, utilized in this manner, are generally applauded by American authorities. Again, since the percentage of hospital confinements in some parts of the British Isles may be as low as 50 per cent, a thorough discussion of the technique of home delivery together with a list of the necessary medical equipment is duly noted.

Excellent chapters on vital statistics and reproduction, family planning, psychologic aspects of obstetrics and gynecology, and a brief discussion of medicolegal considerations add flavor to the otherwise standard material.

The book is designed for student rather than practicing specialist consumption.

RICHARD S. MUNFORD, M.D.

onable  
ent. In  
gen is  
stage  
in this  
erican  
f hos  
ritish  
h dia  
gethes  
ent is  
repro  
ets of  
ssion  
o the  
than  
M.D.